The Milbank Memorial Fund

QUARTERLY

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IN THIS ISSUE

THE substitution of highly refined food products for natural foodstuffs was one important development of the modern industrial era which brought an ever increasing number of the population into cities. Storage and distribution of foods required its preservation for long periods and this resulted in many modifications in the modern diet. The science of nutrition now has established the fact that nutrients essential to human nutrition have been removed from the refined foods to which the public taste has become educated. The question of restoring these essential elements to foods becomes important, therefore, and was discussed by a group of experts at a symposium on Fortification of Foods at the recent annual meeting of the Institute of Nutrition. The five papers read at this symposium include: "Basic Nutrition Principles Involved in the Fortification of Foods," by Dr. Agnes Fay Morgan of the University of California; "Fortification of Foods in a General Program for Better Nutrition," by Dr. Lydia J. Roberts of the University of Chicago; "Public Health Aspects of Fortification of Foods," by Dr. W. H. Sebrell of the United States Public Health Service; "Governmental Control Problems in the Fortification of Foods," by Dr. E. M. Nelson of the United States Department of Agriculture, and "Viewpoints of the Food Industries Regarding Fortification," by Alonzo E. Taylor, Director Emeritus of the Food Research Institute of Stanford University.

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Familial incidence of diseases has long been of interest and has an important bearing on our understanding of the epidemiology of a disease. A higher frequency of any disease among related groups is of significance in the application of preventive measures, whether that incidence arises from infection as a result of familial contact or is due to inherited pre-

disposition to the disease. The study "Further Observations on the Occurrence of Rheumatic Manifestations in the Families of Rheumatic Patients" by Ross L. Gauld, Antonio Ciocco, and Frances E. M. Read is a significant contribution to the epidemiology of rheumatic disease.

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One of the significant contributions of epidemiological research in tuberculosis has been to demonstrate that the risk of disease and death is highly concentrated within an exposed group, those in familial contact with the disease. A natural corollary to this finding is further investigation as to the full effect of tuberculosis upon the growth of the family unit. Such an investigation is reported upon by Miss Jean Downes of the Fund's staff in the article "The Effect of Tuberculosis upon the Size of Family." The broad indications of the study are that, due to a lower fertility among the tuberculous operating in conjunction with an excessively high mortality among their offspring, the tuberculous family tends to be eliminated much more rapidly than do families drawn from the general population. This has probably been a factor contributing to the decline in the tuberculosis death rate which has been occurring over a long period of years and in the future the size of the family unit among the tuberculous will play an increasingly important part in the decline in mortality from the disease.

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Past studies of variations in fertility according to occupational class have been largely restricted to married women classified by the husband's occupation. This procedure operates toward holding constant the factor of group differences in proportions married. In "Differential Reproduction in England" Dr. Christopher Tietze presents birth rates by occupational class when the full influence of variations in marriage frequencies is retained. His technique is that of relating births to males instead of females, the advantage being that unmarried and married males can with equal readiness be divided into occupational groups. The findings from the English material emphasize the need for learning more about the bearing of marriage frequencies on class differences in reproduction in this country. An analysis directed toward this end is now being made

under the joint auspices of the United States Public Health Service and the Milbank Memorial Fund. Dr. Tietze's paper also contains some figures on differences in infant mortality rates according to occupational status of the father.

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To the extent that increased practice of family limitation has played a part, the general decline in fertility rates signifies progressively fewer involuntary births. In view of the lack of similarity in rates of decline in fertility in the various age groups, Dr. Henry S. Shryock, Jr. has posed for examination the hypothesis that a relatively fixed "level or plateau" in fertility rates will be apparent in some age groups earlier than in others. Under the title "Trends in Age-Specific Fertility Rates," he presents a critical analysis of this hypothesis, using several series of historical data for different countries of the Western World. The data are set up to show average annual rates of change in age-specific birth rates during stated periods since the World War. Particular attention is devoted to trends during the most recent period, that of partial economic recovery beginning in 1934.

FORTIFICATION OF FOODS WITH VITAMINS AND MINERALS

THE BASIC NUTRITION PRINCIPLES

AGNES FAY MORGAN, PH.D.[1]

SINCE there are many diverse aspects to the topic of our discussion, it is necessary that I define at the beginning my interpretation of the phase assigned to me. I assume that it is my duty to discuss (1) any advantages to be derived from the provision of increased amounts of vitamins and minerals in the common diet, and (2) any disadvantages or dangers resulting from such increases. Further, I assume that the probable need for additions to the usual American diet will be considered in detail by another speaker and that my concern is largely with the effects, both good and bad, of excesses.

There are two questions to be answered in regard to possible *advantages* to be gained by the artificial addition of vitamins and minerals to foods: (a) Is it desirable to add these substances to processed foods so as to restore these foods to their natural composition? and (b) Is it desirable to add an excess of the chemically pure substances to both processed and natural foods?

The answer to the first question obviously is yes, if we accept the doctrine that man has survived by functioning best in the environment in which he has been evolved. Human experience and animal experimentation have demonstrated beyond doubt that *whole fresh* foods properly selected can support abundant health without addition or sophistication. But the exigencies and refinements of modern civilization have decreed that most urban, and to some extent rural, populations must subsist upon foods which are neither whole nor fresh, however wholesome and palatable they may ap-

[1] University of California, Berkeley.

pear to be. If the exact qualities of whole fresh food can be restored to the sophisticated and preserved foods upon which we must largely depend, the return to nature so often advocated will have been accomplished. But the indiscriminate acceptance of foods merely because they are whole and fresh will not ensure good nutrition, since a vast profusion of such foods, of widely different values, is now produced by our over-adequate scientific agriculture. Although good, fair, and poor diets may still be selected by haphazard choices, the chance of nutritive disaster would be greatly decreased if only whole fresh food were used.

Perhaps no one would dispute seriously the desirability of making available to all a wide choice of foods fully equivalent to their own whole fresh original state. This means for example the restoration to canned and dried foods of the vitamin B_1 and vitamin C content lost in processing, the addition of both these vitamins and vitamins A, the B_2 complex, and the ash constituents of sugar cane and sugar beets to the pure sucrose made from them and to confections containing much sucrose; the water soluble vitamins, particularly B_1, to all wines and liquors; the vitamins and minerals removed from flours and cereals in milling, and the intangibles lost in cold storage and freezing of all foods, but particularly fruits and vegetables. In this connection we must consider also whether separated fats of both animal and vegetable origin can be considered natural foods and whether muscle meats eaten in the present preponderance over organ meat can be considered whole foods. Clearly, full restoration of the qualities of *wholeness* and *freshness* to our civilized fare cannot be considered an easy or simple task. Indeed, in the present state of our knowledge of nutritive requirements, it may be impossible of accomplishment.

But certain plain deficiencies are evident and universally acknowledged. Chemically pure sucrose, decorticated and degerminated cereals, canned and dried foods have lost certain important constituents which can be restored in part to them, or which can

be left in them in whole or in part, by modifications of their industrial preparation. Such a change cannot be other than advantageous.

But even though these obvious deficiencies in important groups of foods were amended, the return to whole fresh diet would be only partly accomplished. New indications of the importance of new "little things" in nutrition are published almost daily. The return of some of the better established factors cannot assure us of adequacy until the whole story has been unfolded. Madame Lucie Randoin has expressed our state of uncertainty admirably in a talk made a few years ago about the nutritive properties of "the precious wines of our sweet France." (1)

> "Aside from the transformation of sugar into alcohol, the chemical and physico-chemical composition of the precious liquid, as prepared by nature, should be modified as little as possible. Watch closely that the vital equilibrium of the wine is not disturbed by the process of treatment, that certain necessary substances in the wine are not destroyed" [fragile substances which exist only in minimal doses]; "these must be the principal precepts to be followed by all those who take part in the fabrication of wine" [and one may add of any other food].

The second question must now be considered. Is it desirable to add an *excess* of vitamins and minerals beyond the amounts ordinarily present in whole fresh foods? What evidence is there that "spiking" foods with constituents never fabricated in them by nature is either harmless or advantageous? Should cereals and milk carry vitamin D as excess baggage, yeast and cough drops vitamin A or carotene, whiskey and pork sausage vitamin C? There is no positive answer available at this time. However, some evidence is beginning to appear that latent deficiencies of one vitamin may become frankly evident when an excess of another vitamin is administered. This is what Dr. P. Gyorgy calls the "concurrence of the vitamins." It has appeared, according to Spies and Aring (2), that pellagrins treated with large doses of nicotinic acid develop new symptoms characteristic of vitamin B_1, and other, deficiencies.

Margolis, Margolis, and Smith (3) found that dogs which had been treated with nicotinic acid for blacktongue had markedly increased vitamin B_1 requirement. In our own laboratory rats showing the relatively mild symptoms of the anti-grey hair filtrate factor deficiency develop fatal paralysis accompanied by severe dermatitis when given moderate doses, 1 to 3 mg. daily, of nicotinic acid; Dr. T. H. Jukes reports similar effects in chicks (personal communication) and also an exacerbation of filtrate factor dermatitis on administration of riboflavin (4).

A possible parallel occurs in the phosphorus deficiency which may result from precipitation of insoluble phosphates by the presence of an excess of calcium or iron in foods, particularly in cereals which contain variable amounts of poorly utilized phytin phosphorus. A reciprocal deficiency of calcium or iron may result from the presence of excess phosphate. Certainly unbalanced and uncritically adjusted additions of any of these minerals to foods may result in unexpected deficiencies.

If the calcium added to foods be in the form of one of the calcium phosphates, adequate absorption cannot be expected in all cases, particularly in young children and in elderly persons and others with low gastric hydrochloric acid. If it be in some other form such as gluconate or lactate the danger of phosphate precipitation and deficiency arises.

Perhaps the most dramatic and universal example of this danger is the rickets-producing effect of cow's milk fed to human infants. The absolute and relative excess of calcium over phosphorus provided by this food as compared with the needs and digestive capacity of the infant often results in intestinal precipitation of calcium phosphate and low phosphorus rickets. By treatment with vitamin D in one way or another normal conditions can be reattained in these rachitic children, an expedient probably not contemplated by nature. The provision of cow's milk fortified with vitamin D may be looked on therefore as the necessary and desirable

concomitant of the salvage of infants fed cow's milk instead of their rightful heritage, human milk.

The decline of milk production by mothers in recent years may itself be related to the increasing sophistication of diet, since lactation seems to be peculiarly dependent upon the intake of the B vitamins as well as of certain amino-acids. If a case can be made for the general use of fortified foods and concentrates by any part of the population it can perhaps be made for lactating women. The value of such fortified diet would lie, however, in the protection of the mother and in the maintenance of the milk supply rather than in any great improvement in the vitamin content of the milk. The case for the use of fortified foods by pregnant women seems to be less complete.

The second part of our problem may now be approached. Is there *danger* from *excesses* of vitamins and minerals? I have already pointed out the danger of loss of calcium, iron, and possibly other minerals through the presence of excess phosphate and of loss of phosphate through excess of the minerals. Actual poisoning from large doses of copper, ferrous salts, or manganese and iodides may of course occur. Damage to the kidneys may result from excessive calcium or phosphate ingestion either with or without vitamin D.

There can be no doubt that vitamin D in excess is toxic and that the margin between therapeutic and toxic doses is variable and influenced by unknown individual factors, possibly related to parathyroid activity, as well as by the calcium and phosphorus composition of the diet, insolation, type of vitamin D used, and level of intake of certain other vitamins. That an excess is cumulative and rather prolonged in after effects has been established. The use of a large variety of irradiated and vitamin D fortified foods or fish-liver oil concentrates supplemented by sun lamp and sunlight exposure is fraught with danger, particularly for the very young and the elderly. We have seen with rats, dogs, and guinea pigs (5) that the younger the animal the greater the damage done by a given excess

of vitamin D and that elderly animals fail much more rapidly under constant mild hypervitaminosis than do young adults.

The observations of Jeans and Stearns (11) that infants given 1,800 units of vitamin D daily have linear growth rates definitely retarded below that of similar infants receiving 400 units daily provide a sharp reminder that the therapeutic and toxic levels of vitamin D are not as widely separated for the human as for the rat.

Some reports of damage due to excess vitamin A and carotene have come from European laboratories but these have not been confirmed. In fact Davies and Moore (6) and Bell, Gregory, and Drummond (7) as well as Vedder and Rosenberg (8) have shown conclusively that enormous doses of these substances cause no damage.

This reassurance cannot be offered however for fish-liver oils and concentrates as a whole. There is an undoubtedly harmful effect, involving muscular dystrophy, accruing from these oils to certain herbivora and in somewhat less degree to other species, such as the rat. Many claims for counter-action of this fact by the water-soluble vitamins (Burack and Zimmerman) (9), (7), have been made but no satisfactory explanation is as yet available. Certainly caution in the use of fish-liver oils should be exerted until this matter is cleared up.

No very obvious effort has yet been made to exploit vitamin E concentrates and danger from excess of this substance has not been demonstrated. However wheat germ oil which is often used as a concentrated source of vitamin E has been accused by Rowntree and co-workers (10) of promoting tumors in the rat. Several other laboratories have attempted but failed to reproduce this condition. Further study of the possible relation of vitamin E to tumor production is indicated before concentrates can be recommended for even general therapeutic use.

Nicotinic acid amide is the only one of the water-soluble group of vitamins which may be looked on as a toxic hazard when taken

in excess. Several clinical reports have already shown plainly that transient and apparently harmless unpleasant effects are experienced by nearly all persons who take doses which are not in excess of the therapeutic range. Massive doses are said to produce symptoms in rats similar to those of curare poisoning. Certainly this is an accessory food factor which must be used with caution because of its inherent toxicity as well as the concurrence of other vitamin deficiencies which it may entail.

Nevertheless the use of fortified foods and vitamin concentrates can be justified probably in all restricted or reduced diets, especially those in use for long periods of time and in persons with gastrointestinal derangements. These "special purpose" foods are unquestionably needed in reducing diets, diets for nephritic and cardiac conditions, for alcoholics, for old age, for peptic ulcer, diabetes, in preparation for surgery, and for all the deficiency conditions. Such foods should be available when needed but should hardly be forced upon the attention of the general public. A debatable case in point is that of iodized table salt, of unquestioned value to the iodine-deficient people in regions of endemic goiter but even in such regions a possible danger to those with adenomatous thyroids.

Vitamin B-reinforced whiskey or beer might be an excellent product to recommend to the confirmed alcoholic who could afford it but should hardly be advertised to the general public by high-minded manufacturers as a "health food." Ascorbic acid instead of orange juice may be prescribed for diabetics or cachectic infants but even for them can hardly be the preferred choice if the vitamin P claims of Szent-Gyorgyi are accepted. In our own laboratory we have had convincing evidence recently with the help of synthetic diets for guinea pigs, adequate except as to vitamins C and P, that both orange and tomato juices carry something which is indispensable in addition to ascorbic acid.

Vitamin-poor foods which are "perversely" used, such as sugar, candy, polished rice, white bread and the sugary foods made with

white flour, all alcoholic drinks, and all artificial soft drinks form a group which might be considered among "special" foods, indispensable to the wretches addicted to them because of habit, ignorance, or poverty and therefore candidates for fortification, enforced or voluntary. If chemical fortification is cheaper than return to the manufacture and distribution of the whole fresh precursors then let us have thiamin and the B_2 complex added to sugar, white flour, polished rice, liquors and soft drinks, ascorbic acid to the beverages and jellies, but without fanfare and exorbitant profit. A better solution might come from education of the consumer about the impoverished state of these foods, but the people learn slowly and the results of subacute deficiencies are not dramatically evident nor associated in the popular mind with their true causes. Perhaps we must go through an era of exploitation of fortification of some of these foods in order to speed up the learning process.

This raises the question of the choice between rational selection of *whole fresh* foods and the use of the foods that we like, or are accustomed to, or can get, made safe with a daily supplement of vitamin and mineral concentrates from the drugstore. Many intelligent people who can afford it, are now following the latter course. This is, I believe, a dangerous, expensive, and a passing phase in our struggle toward optimum nutrition for all the people. Karl Thomas (12) wrote once about amino acids the following questions: "1. What amino acids must be present in the food? 2. How much must we have of each? 3. To what purpose?" If we apply this to vitamins we can see that we have only a partial answer to the first question, a bare beginning as to the second, only a suspicion as to the third. "Proceed with caution" must remain the watchword still.

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FORTIFICATION IN A GENERAL PROGRAM FOR BETTER NUTRITION

LYDIA J. ROBERTS, PH.D.[1]

WHAT is the significance of fortification in a general program for better nutrition? To answer this question satisfactorily would require more accurate knowledge than we now possess of the present dietary habits and the nutritional status of the people of the country. From the evidence which is available, however, it seems impossible to escape the conclusion that the diets of our people at their best have no great margin of safety, and that a significantly large proportion of them, especially in the lowest income groups, are deficient in one or more of the dietary essentials. This is strikingly illustrated in the study recently reported by Stiebeling and Phipard of 4,000 families in various sections of the United States (1). Outstanding among the deficiencies, in the judgment of the writer, are those of calcium and vitamin B_1, and in the southern states also the pellagra preventive factor, or factors. Iron, vitamins C and D, and other fractions of the B complex are also questionable and in none of the constituents is there any generous margin of safety. I shall present some evidence for calcium, and vitamin B_1, and for the marginal nature of the diet as a whole.

CALCIUM

The best index of the calcium intake is found in the consumption of milk, for this is the only liberal source of this constituent in the American diet. Although there has been a steady increase in the per capita consumption of milk during the past few decades it is still far from adequate. This is borne out by a study made in 1934, through the cooperation of the United States Department of Agriculture and twelve national women's organizations, of the milk

[1] From the Department of Home Economics, The University of Chicago.

consumption of 28,966 families in fifty-nine cities in all sections of the country (2). The average milk consumed by these families was 2.44 quarts per person per week, or less than three-fourths of a pint per day. Moreover, 459 families had no milk at all and two-thirds of the families had less than two quarts per person per week, or less than 0.6 pint per day. As might be expected, the per capita consumption was lower in the southern states, in families with the largest number of children, and in the lower income groups. This study is typical of many others which show that the consumption of milk by thousands of families comes nowhere near an adequate standard for building and maintaining strong bodies.

We are beginning, moreover, to accumulate some rather clear-cut evidence of widespread calcium deficiencies, even in adults. A recent editorial in the *Journal of the American Medical Association* (3) calls attention to the fact that generalized osteoporosis is common in adults, especially after fifty years of age. Orthopedic physicians report that many adult patients show extreme demineralization of the skeleton as revealed by roentgenogram, and they believe this to be the unrecognized cause of many of the ills and deformities of the elderly, such as the general loss of vigor, pathologic fractures of the vertebrae, extreme bowing of the spine which is often sufficient to shorten the stature materially, and a variety of other ailments. They believe this demineralization can be explained by a prolonged negative calcium balance over a period of years, due to the lack of calcium in the diet, or to insufficient vitamin D to favor its utilization. Ghormley (4) has shown that in many such cases improvement can be effected by a calcium high diet or a calcium salt with vitamin D. There seems, then, little doubt that the diets of a considerable number of our population are suboptimal for calcium and possibly also for vitamin D.

EVIDENCE FOR VITAMIN B_1

What evidence do we have for our belief that there is a shortage of vitamin B_1 in the American diet? First of all, we may cite the

difficulty encountered when one sets out to plan a diet which meets the standards for vitamin B_1 which have been proposed. Estimates of the B_1 requirement range from 200 to 600 international units. It is possible by the inclusion of liberal amounts of protective foods to bring the B_1 up to the minimum standards, but difficult, if not impossible, to reach the optimal ones and still include the quantities of white bread, sweets, and other refined foods to which people are accustomed. It is significant to note how the modern diet differs in this respect from that consumed by the average American one hundred years ago. Joliffe (5) has made an interesting analysis of this problem. He points out that not only has the consumption of cereals decreased during the last century but modern methods of milling have greatly reduced the content of B_1. In 1840 cereals furnished 32 per cent of the calories in the diet; they now constitute but 24 to 25 per cent. Whereas the process of milling one hundred years ago recovered from 85 to 90 per cent of the grain as flour and this contained 75 per cent of the original B_1, modern methods utilize but 70 to 75 per cent of the grain and the resulting flour contains but 10 per cent of the B_1.

The increased consumption of sugar is another factor that has helped to reduce the B_1 intake. The per capita consumption of sugar which in 1821 was about eight pounds per year has risen to well over one hundred pounds at the present time. (Some estimates give 119 pounds.) This furnishes about 18 per cent of the total calories, in contrast to 1.3 per cent in 1821. For a 3,000 calorie diet this amounts to 540 calories from a food totally devoid of any other dietary essentials. Joliffe points out that these two foods—cereals and sugar—comprise about 50 per cent of the diet. Thus a 50 per cent fraction of the calories which in the diet of 1840 provided 600 international units of B_1 has been replaced by one furnishing but 50 international units. The modern diet is, therefore, poorer in B_1 by 500 to 550 units. Baker, Wright, and Drummond (6) have shown similar changes in the English diet. From calculations of

actual dietaries they find that the charity diets of 1838 provided 1,000 to 1,200 international units of B_1, while those of the well-to-do Englishman today contain about 450 international units and those of the lower income groups only about 290 international units.

Offsetting the loss of B_1 from cereals, of course, has been the increase in the consumption of milk, fruits, and vegetables, but as Joliffe has shown, it would require very large quantities of these to make up for the 500 or more international units lost from the cereal source. It is true, of course, that the B_1 provided by these earlier diets may be in excess of that actually required. This we shall not know until the requirements for optimal nutrition have been eventually determined. But it is, in any case, significant that our diets today are very poor in B_1 as compared with those of our forbears.

Evidence is fast accumulating, moreover, that shows that B_1 deficiency is more common in this country than has been generally assumed. Strauss (7), Joliffe (5), and others have presented an impressive amount of evidence to show that beriberi in characteristic form is endemic in many parts of the United States, but that its presence is concealed by its diagnosis under many different names. It is their conviction that various forms of polyneuritis which are common among women during gestation, in persons with chronic gastro-intestinal disorders, in diabetes, in hyperthyroidism, and in alcoholism, and many other conditions, are all true beriberi caused by a relative lack of B_1 in the diet. They have shown that the calorie/vitamin ratio of the diets in such cases is below the 2.7 which Cowgill considers the level below which beriberi appears, and that the increase in vitamin B_1 with no other change in their diet will effect a cure. Spies (8) states that a B_1 deficiency is also almost invariably present in cases of pellagra, since the nervous manifestations do not clear up on treatment with nicotinic acid alone but do respond to thiamin therapy.

It seems obvious, then, that a true B_1 deficiency exists in a significant but unknown degree. When a deficiency disease is endemic,

moreover, a much larger proportion of the people are probably on the borderline or below and would be shown to be deficient if adequate methods of diagnosing subclinical cases were available.

NATURE OF THE AMERICAN DIET

Time does not permit the presentation of evidence of possible deficiencies in other dietary factors. It is pertinent, however, to call attention to the marginal nature of the American diet as a whole. Some indication of this is afforded by a comparison of three types of diets, as given in Table 1. One consists solely of natural food stuffs; that is, every item in the diet contributes its original quota of minerals and vitamins. One termed the "Good American" contains the amounts of the protective foods—milk, eggs, fruits, vegetables, meat, potatoes, and whole-grain cereals—commonly included by nutritionists in their rule-of-thumb formula to insure adequacy; the rest of the diet is made up from cereals, sugars, fats, and bread which supply little but calories and some protein. The third, termed

Table 1. Foods included in three types of diet.

Natural Foods			"Good American" Diet			"Poor American" Diet		
Food	Amt.	Cal.	Food	Amt.	Cal.	Food	Amt.	Cal.
Milk	1 qt.	672	Milk	1 pt.	336	Milk	1 c.	168
Egg	5/7	50	Egg	1	75	Egg	¼	20
Potato	3 sr.	400	Potato	1 sr.	150	Potato	1 sr.	150
Beans	⅓ sr.	50	Meat	1 sr.	150	Meat	2 sr.	300
Cabbage	4 sr.	75	Orange	1	70	Orange	¼ sr.	20
Apples	2	150	Apple	1	80	Apple	1 sm.	50
Whole Cereal		1,000	St. Beans	1 sr.	25	Lettuce	1 sr.	10
Molasses		200	Lettuce	1 sr.	15			
Fat	Butter / Other	400	Whole Cereal	1 sr.	100			
Sub-Total					1,001			718
			White Cereal		1,000	White Cereal		1,100
			Sugar		500	Sugar		700
			Fat	Butter / Other	500	Fat	Butter / Other	500
Total		2,997			3,001			3,018

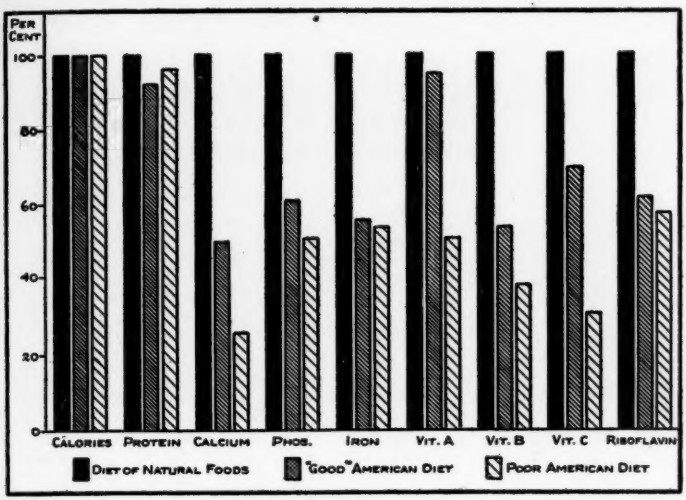

Fig. 1. Comparison of the nutritive value of three diets, natural foods, "good" American diet, and "poor" American diet.

the "Poor American," consists largely of meat, potatoes, sugar, white bread, and other refined cereal products with only meager amounts of the protective foods. The amounts indicated in the table are, of course, arbitrary ones but they serve adequately for purposes of comparison. The nutrients provided by these three diets are graphically compared in Figure 1. For each constituent the diet of natural foods is used as 100 per cent; the others are compared with it.

It is seen that even the so-called "Good American" diet falls a long way below the diet of natural food stuffs in many respects. This is explained by the fact that the protective foods constitute but 1,000 calories, or one-third of the total calories, and this fraction is depended upon to carry all the minerals and vitamins; the remaining two-thirds of the calories are derived from white bread and other refined cereal products, sugar, and fat, which are practically devoid of these constituents. It should be pointed out in this con-

nection that this food plan is not advocated by nutritionists as an ideal. It is merely set up as one way of assuring a minimum of adequacy in dietary essentials even while conceding to the popular demand for a diet containing a large proportion of refined foods. If this rule-of-thumb method is followed literally, moreover, the diet will usually meet at least the minimum dietary standards, but it has no generous margin of safety such as is afforded by the diet of natural foods.

Unfortunately, such evidence as is available indicates that relatively few people regularly have diets even quite as good as this. A considerable proportion of our population are subsisting on diets more like the one we have termed the "Poor American." As may be seen in the chart, this diet measures up to the one just discussed only in calories and protein, and is definitely below it—and consequently below even the minimum dietary standards—in most respects.

METHODS OF IMPROVING THE DIET

It is readily apparent that the American diet needs improving. How can this be accomplished? There can be no question that the ideal solution would be to bring about the needed improvement by increasing the use of the protective foods, and by returning to some extent at least to a greater use of some of the less-highly milled cereal products and other refined foods. Programs of popular education should, moreover, be directed toward this end. Desirable as this end is, however, it can be accomplished only by a long and vigorous program of popular education, and we are faced with the immediate need to do something to better the nutritional status of our people. The line of least resistance, therefore, might be to add to certain basic foods which are consumed in largest amounts by the mass of the people the dietary essentials that have been shown to be most lacking in the usual diet, so that no matter what they eat, they would "willy-nilly" have an automatic source of the dietary essentials. This method has already been used with iodized salt and

with vitamin D milk, and the advisability of extending this plan to other foods is being seriously considered.

If this procedure is to be used, then highly refined products, especially flours, would seemingly head the list of foods to be fortified. I have presented evidence, for example, that our diets are submarginal in vitamin B_1. The suggestion has been made that some potent source of B_1, such as a highly potent yeast or crystalline thiamin be added to all flour and milled cereals so that every cereal product that people ate—bread, macaroni, pastries—would automatically contain it. Would this be in the interests of better nutrition? In my judgment, it probably would, *if* it could be done at no additional cost to the consumer—for the lower income groups are the ones that most need to have it provided in this way—and *if* all advertising for the resulting product could be properly controlled. Similarly the addition of yeast or of nicotinic acid, as well as B_1—and possibly also riboflavin—to the cornmeal and flour used in the sections of the country where pellagra is endemic might be an advantage to the general health, providing the entire fortification program could be under medical direction so that it would be properly safeguarded.

The chief danger I see in such fortification procedure is that it might tend to give a false sense of confidence that all the deficiencies of refined cereals have been overcome. It must be remembered that cereals have lost far more than just thiamin or any other single vitamin in the process of milling and that some of these other factors may be equally important in human nutrition. Figure 2 has been prepared to illustrate this point. It shows the nutrients provided by 750 calories of cereal—the amount statistics indicate to be the present average per capita consumption—when used as whole-meal flour, and as white flour. The solid bar in each pair represents the amount of the nutrient in the whole-meal expressed as 100 per cent; the hatched bar indicates the relative amount in the white flour. It is seen that the amount of protein is essentially the same in the two types of flour but that in all other materials the white flour

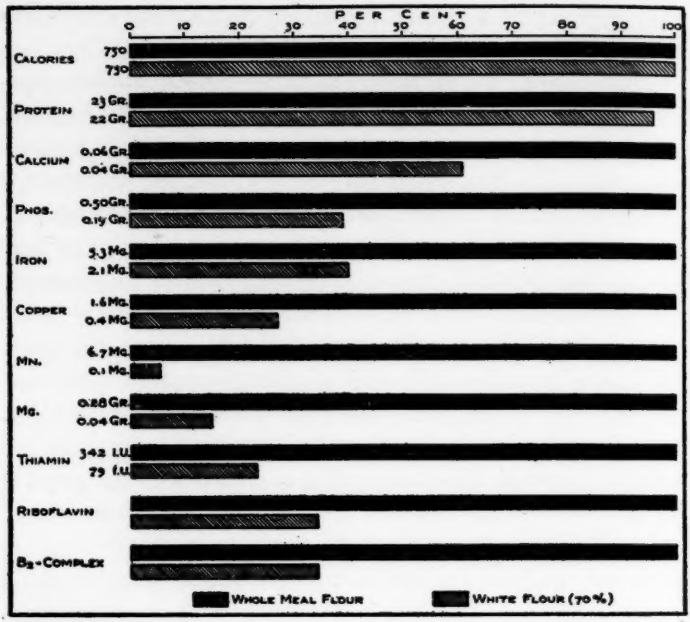
PER CENT
0 10 20 30 40 50 60 70 80 90 100
CALORIES 730 730
PROTEIN 23 GR. 22 GR.
CALCIUM 0.06 GR. 0.04 GR.
PHOS. 0.50 GR. 0.15 GR.
IRON 5.3 MG. 2.1 MG.
COPPER 1.6 MG. 0.4 MG.
MN. 6.7 MG. 0.1 MG.
MG. 0.28 GR. 0.04 GR.
THIAMIN 342 I.U. 79 I.U.
RIBOFLAVIN
B_2-COMPLEX
WHOLE MEAL FLOUR
WHITE FLOUR (70%)

Fig. 2. Dietary essentials furnished by 750 calories of cereal when eaten as whole meal flour and as white flour.

is definitely poorer. In terms of the whole-meal flour it has lost one-third of its calcium, two-thirds of the iron, three-fourths of the copper and thiamin, and almost all of the manganese and magnesium. According to the data given by Copping (9), the loss of flavin and other B_2 factors is from 34 to 80 per cent. The decrease in calcium and phosphorus is probably less significant, but the loss of several milligrams of a well-utilized iron and the other minerals whose values in nutrition are just beginning to be appreciated, as well as other fractions of the B complex, may be a matter of importance.

While the supplementation of cereals with one or two vitamins then would help to remove some of the deficiencies in the American

diet, the fortified product will be far from the equivalent of the original cereal. If the plan is accepted, the exploitation of the product should be so safeguarded as not to give the impression that all the deficiencies of the cereal have been overcome. It would seem desirable indeed that other constituents which cereals are normally expected to contribute should also be added, or more properly, retained in the product by the process of milling.

If fortification is accepted as an automatic method of improving the diet, the safest procedure would appear to be along the line of fortification of natural carriers. Addition of iodine to salt, and the various B factors to cereals are of this nature. I would personally favor also the addition of vitamin A to all butter substitutes to at least the amounts found in a good grade of summer butter, so that the poor man's diet would contain a more liberal supply of this vitamin. Further than this I am not prepared to go at the present time.

To sum up, I believe that, if it could be properly safeguarded, a judicious use of fortification might be of decided benefit in a general program for better nutrition. If undertaken, leadership in the movement should be given by physicians and specialists in nutrition who should work out carefully considered policies as to the foods which should be fortified, the materials to be added, and the absolute and relative amounts to be used, and who should also direct the distribution and promulgation of the fortified products.

Whatever is done in the way of fortification, however, must always be regarded as merely a supplementary or "first-aid" measure. Our major emphasis should be directed toward the improvement of the diet along the lines already indicated as desirable. We must remember that the last word has not yet been spoken in respect to nutrition and that there may still be many essential dietary factors of which we are as yet unaware. We can not, therefore, trust solely to fortification, but must still put our major faith in a varied diet containing generous amounts of natural foods.

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THE PUBLIC HEALTH ASPECTS OF THE FORTIFICATION OF FOODS WITH VITAMINS AND MINERALS

W. H. SEBRELL, M.D.[1]

THE question of the desirability of fortifying foods with vitamins and minerals from the point of view of public health or preventive medicine is a most important one because the principal, if not the only possible reason for doing this is the prevention of deficiency diseases. I can see no other valid reason for even considering the question. By no stretch of the imagination can such a procedure be considered desirable in the treatment of disease. Surely we can all agree that the treatment of the vitamin deficiency diseases can be most satisfactorily carried out by the use of concentrated vitamin preparations properly administered in the hands of qualified physicians. The one legitimate reason, then, for fortifying foods with vitamins and minerals is to prevent disease, and it is from this point of view that we should examine the problem. There are several important questions we should ask ourselves in this connection—such as—is it practical, is it economical, is it efficient, is it safe, and, above all, is it the best method for us to use in attacking the problem of the prevention of our deficiency diseases?

At this point I want to make a special exception for the case of the fortification of milk with vitamin D for children in certain areas. Because of our inability properly to expose the skin of infants to direct sunlight at all seasons of the year, increasing the vitamin D content of cow's milk for infants in some areas appears to be one of the most satisfactory means of preventing rickets. Although this procedure is subject to many of the objections to fortifying any

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food, the fact that it enables us to offer vitamin D in a form in which it will be given to infants more than offsets all the arguments against it. I therefore feel that this is a special case which in itself makes it undesirable to add vitamin D to other foods.

Let us then try to answer some of the questions I mentioned a moment ago in regard to fortifying all other foods with vitamins and minerals.

In the study of preventive medicine we are taught that certain basic information must be available before an effective effort can be made to prevent any disease. It is fundamental that we must know first whether the disease in question is present and, if so, to what extent. We also must know where the disease is located, what its cause is, and what part of the population is most seriously affected by it. Without such information one cannot even begin to adequately prevent and control any disease, and it is the lack of just such information that has seriously handicapped us in our efforts to prevent the deficiency diseases. We do have some of the necessary information. We know that dietary deficiency diseases exist in the United States. We have plenty of rickets, scurvy, pellagra, and nutritional anemia, among other things. However, we do not know to what extent any of these diseases occur, and we do not know where the cases are located. In addition, there is absolutely no adequate information available on the prevalence of the so-called subclinical stages of the various vitamin deficiency diseases. The fact that we cannot state with accuracy either the number of cases of vitamin deficiency diseases occurring each year or where they occur might erroneously lead one to the conclusion that this constitutes a good reason for trying to prevent these diseases by fortifying commonly-used foods with vitamins and minerals. This is a method similar to that condemned years ago by the medical profession as the "shot-gun" method of treatment. By scattering your vitamin shots over the entire population you hope that a few pellets will hit the individuals needing them. Unfortunately, this method does not

cover the possibility of your pointing the gun in the wrong direction and missing the bird entirely, because by fortifying foods with vitamins you increase their cost and move them farther than ever from the reach of the population group needing them most seriously.

Although, as I have indicated, our accurate knowledge about the occurrence of the deficiency diseases is meager, we do have much information which leads us to make worth-while estimates about conditions. For example, no one will question that our pellagra problem is primarily one of the southern United States, while our rickets is a more serious problem in our northern and northeastern sections. Therefore, we can make a crude geographic location of two of our most serious vitamin deficiency diseases. But much more important than this is the evidence that has accumulated on the relationship between the deficiency diseases and income. Goldberger, many years ago, showed a direct relationship between income and the incidence of pellagra in the South, and more recently many studies, but especially the extensive work of Stiebeling and her associates in the United States, have shown very clearly that the lower the income the greater is the probability that a family will obtain an inadequate diet. I think we are justified, therefore, in locating our public health problem in the prevention of the dietary deficiency diseases in that section of the population with the lowest income in any given area.

Now, then, since our purpose in fortifying foods with vitamins is the prevention of these diseases, it is certainly this low-income group that needs our vitamin-fortified foods, and what are we doing? Actually, we are making it more difficult for them to obtain these foods for there can be no question but that the addition of vitamins to foods will increase the cost, and every penny added to the cost of food simply places it farther than ever away from this lowest income group which is receiving a deficient diet because the cost of the unfortified foods is already beyond their means. We

should be devoting our efforts to decreasing the cost of food for this group rather than increasing it.

The fortification of foods with vitamins and minerals, then, in all probability would not only fail to be of any material value in the prevention of the deficiency diseases by not reaching the population group in greatest need of them, but it also would be an economically wasteful procedure, since it unnecessarily increases the cost of these foods to people whose diet is already adequate and who might be persuaded to buy these fortified foods although not needing them.

I have already indicated that a procedure such as this must be regarded as bad preventive medical practice, but there are still other points to be considered. It is becoming increasingly clear that multiple deficiency diseases are the rule rather than the exception. A patient suffering from, say, pellagra, rickets, or scurvy, is quite likely to have symptoms of some other deficiency at the same time. There is really no reason to expect an inadequate diet to be deficient in only one respect. Those of you who have had occasion to study deficient diets as they are found in the field no doubt wonder how some people have escaped having two or three deficiency diseases simultaneously. This situation raises the question of which vitamins and minerals shall we use to fortify foods—if we are going to use any? And this in turn raises a whole host of unanswerable questions. How much shall we add? To what foods shall we add them? Shall we add some to one food and some to another? How will we be able to tell when we have the right amount of the right combination? No one food can have a monopoly and we can expect a variety of foods to be fortified if this procedure is encouraged. Yet, we certainly don't want our meals supercharged with a load of vitamins A to P and minerals aluminum to zirconium. This would not only be unnecessary and wasteful, but we might even become so big and strong and vigorous and youthful that we might want to start a war with somebody. Seriously, though, we should not dismiss too lightly the possibility of harm from overdoses of

vitamins. Although there is no evidence of immediate harm from relatively large excesses, long-time experiments over several generations have not been carried out, and we should also keep in mind the observations of McCay and his associates that animals that grow to maturity slowly live longer than those that mature rapidly.

I think we can safely say that there are so many unknown factors involved in both the question of what to add and how much of each to use in fortifying a food that it would be prudent not to undertake it at this time as a public health measure.

Finally, to me it does seem a little ridiculous to take a natural foodstuff in which the vitamins and minerals have been placed by nature, submit this foodstuff to a refining process which removes them, and then add them back to the refined product at an increased cost. Yet this seems to be the thing that is being proposed. If this is the object, why not follow the cheaper, more sensible, and nutritionally more desirable procedure of simply using the unrefined, or at the most, slightly refined natural food?

Unfortunately, we must face the existing situation that certain refined foods are our cheapest sources of food energy and that there is a public desire for white foods with a background which is difficult to break down, particularly among the uneducated. Instead of attempting to correct the situation by fortifying these foods at increased cost, it seems better procedure to me to try to build up an association between good health and a colorful diet. A diet that has a lot of red, yellow, orange, and green color from natural foods is not likely to be deficient in minerals or vitamins.

It is granted that the use of some refined foods is desirable, but it is indeed an anomaly to find that there is little or no difference in the retail price between some refined foods and the same food unrefined. For example, brown rice retails in Washington, D. C., for the same price as polished rice.

I do not believe that our deficiency disease problem will ever be solved by fortifying foods, but it can be solved by education and

by making available to all classes the widest variety of natural, unrefined foods at the lowest possible prices.

Stiebeling's investigations show that even today in the groups spending small sums of money for food a small proportion of the families, by wise spending, are able to obtain an adequate diet. This in itself shows the solution to the problem. I, for one, would certainly never advocate among that group of people the spending of one extra dime for a refined food fortified with vitamins and minerals.

Let us consider for a moment the reasons why we have deficiency diseases. Broadly, they are our needless refining of foods and the inability of our lowest economic groups to obtain a varied diet, either through ignorance or economic causes.

Let us take an example of a public health problem in this field. It is common experience for the southern health officer to find himself confronted with a considerable number of cases of pellagra. This is a problem in treatment which is handled by the local physicians and the health officer in the best way they can. The question here is: How can this health officer prevent future outbreaks in his community? Should he introduce some food fortified with nicotinic acid or should he attempt to get his population to eat a more varied diet which will afford protection against pellagra? In my mind there is no question as to the preference for the latter method, because by so doing he also prevents the other vitamin deficiency diseases. Or, to take as another example, the recent attention which has been given to scurvy in Maine. The health officer, in meeting the situation, rightly chose to attempt to obtain a supply of citrus fruits and encourage their use rather than use food fortified with ascorbic acid.

Unfortunately, there seems to be some quirk in most of us that makes us want to take a pill. There is something rather final and satisfying after swallowing a pill. After it passes that final obstruction on the way down we are inclined to mentally compose our-

selves to await a miraculous and completely curative result. I am afraid there is much of that psychology behind the fortification of foods with vitamins. I am afraid also that we are beginning to look on food and eating more as a duty than a pleasure. We are so accustomed to the association of illness with unpleasant medicine that it seems difficult to even put across the idea that here is a whole group of diseases that require no pills, no unpleasantness to prevent, and that their prevention can actually be made a most agreeable occupation or even recreation. But this is too simple. I am reminded of the story current down South about the negro who, on being given his first package of debittered brewers yeast, returned to the health department and asked for more of the bitter yeast because the new yeast didn't taste strong enough. The association between disease and medicine is so firmly fixed in the mind of the public that one is looked on with a certain degree of suspicion if you say that all the treatment that is necessary is a variety of nice, fresh vegetables, eggs, meats, milk, and so forth. As a public health measure, I think we should actively oppose the fortification of foods with vitamins and, instead, we should do all in our power to destroy this misconception in the public mind that such things are necessary to prevent the deficiency diseases.

GOVERNMENTAL CONTROL PROBLEMS IN THE FORTIFICATION OF FOODS WITH VITAMINS AND MINERALS

E. M. NELSON, PH.D.[1]

PRACTICALLY all aspects of the desirability of fortifying foods with vitamins or minerals have long been problems of the Food and Drug Administration. These problems have arisen in considering the propriety of the representations that have been made for various vitamin and mineral preparations. Successful sales promotion of such products depends on the extent to which representations can be made that our dietaries do not supply certain food essentials in adequate amounts. It also depends on the representations made with respect to the nature of the ills or conditions which result from such deficiencies. Much has been published on the subject of nutrition in recent years and quite diverse opinions have been expressed. It is necessary for one to be rather circumspect in arriving at a conclusion, or even an opinion, with respect to the value of minerals and vitamins. If one surveys the literature with a view to determining what possible beneficial effects may be expected by increasing the vitamin or mineral content of our foods, he arrives at one opinion. He arrives at an entirely different opinion with respect to their value if he reviews the literature with a view to determining just how much has been definitely established. I could say that this is due to a paucity of established facts, if I could be generous enough to overlook altogether too many statements in scientific literature that reflect a degree of optimism concerning what vitamins can accomplish that is not fully warranted by experimental or clinical evidence. I would have no difficulty in preparing a brochure consisting practically entirely of quotations from scien-

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tific literature which would appear to form a convincing case for extensive deficiency in our dietaries of vitamin A, B, C, or D, or the minerals calcium, phosphorus, iron, or iodine. It could also be shown that a great many of our ailments are due specifically to a deficiency of each of these substances. There is inadequate information concerning the vitamin content of foods actually consumed. Human requirements for vitamins and minerals are not well established. We do not have satisfactory criteria for determining objectively lesser deficiencies which do not produce definite manifestations of specific diseases. Since we do not have adequate information and a decision must be based on individual judgment, humanitarian principles dictate that if there is a possibility of error, we should not err in a direction which may add further to possible human suffering, because the existence or importance of certain inadequacies of diets are not definitely established.

The legislative acts enforced by the Food and Drug Administration do not prohibit the fortification of foods with vitamins or minerals. They do prohibit unwarranted representations for such products. The responsibility of the Food and Drug Administration with respect to representations for foods is limited to the labeling of goods in interstate commerce or importations. This responsibility is voiced in the Federal Food and Drugs Act of 1906 and the Food, Drug, and Cosmetic Act, which is scheduled to become fully effective on June 25th of this year. Authority for the control of advertising for food, drug, and cosmetic preparations has been conferred upon the Federal Trade Commission by the Wheeler-Lea Act, which was passed in 1938. There are some sections of the Food, Drug, and Cosmetic Act which have an immediate bearing on the subject of this symposium. If it is agreed that from the standpoint of protection of public health there is need for the fortification of foods with vitamins or minerals, then it becomes important to know to what extent existing statutes will be an aid or a hindrance in effective distribution and sale of such products. We also want to

know to what extent it is necessary for manufacturers to give the purchaser helpful information.

The Food, Drug, and Cosmetic Act provides for promulgation of definitions and standards for foods whenever this is deemed to promote honesty and fair dealing in the interest of consumers. Whenever a definition and standard has been adopted for a food, that name must be used on the label, and use of the name for that product signifies that it conforms with the definition and standard. It is obvious that there are many foods which are of natural origin or manufactured by standardized processes, all of which are readily identified by common names, but it is necessary to have legal standards for the composition of such products to prevent their sophistication and to have means of preventing undesirable trade practices.

If a definition and standard has not been promulgated for a food and it is fabricated from two or more ingredients, the law requires that the label bear a list of the ingredients by their common or usual name. It will certainly be a revelation to some people to find out that combinations of simple foods have been represented to be helpful or beneficial in preventing various diseases, improving digestive processes, increasing or reducing weight, increasing attractiveness of the figure, improving the texture of the skin, producing sexual vigor, quieting the nerves, and veritably giving comfort to the soul.

Of further importance is the following section: "A food shall be deemed to be misbranded if it purports to be or is represented for special dietary uses, unless its label bears such information concerning its vitamin, mineral, and other dietary properties as the Secretary determines to be, and by regulations prescribes as, necessary in order fully to inform purchasers as to its value for such uses." There is no similar requirement with respect to informative labeling in the Food and Drugs Act of 1906. Please note that this section is entirely ineffective unless regulations have been prescribed. We are now engaged in drafting such regulations, and we welcome any suggestions with respect to how the intent and purpose of this sec-

tion can best be accomplished. The following procedure must be followed in the promulgation of regulations: The Secretary of Agriculture must give appropriate notice of a hearing, and the notice shall set forth the proposal in general terms and specify the time and place for a hearing to be held not less than thirty days after the date of notice. At the hearing any interested person may be heard in person or by his representative. The hearings are held before a presiding officer appointed by the Secretary. After the hearings have been held, the presiding officer prepares a statement of the proposed findings of fact and proposed regulations, which are subject to review by an appellate court if there is a basis for controversy or if a person can show that he will be adversely affected by the proposed regulations.

There is no definition of the term "food for special dietary uses," but it appears to us that it certainly was the intention to include foods fortified with vitamins or minerals under this section. A provision requiring statements on the label which fully inform the purchaser obviously makes it possible to require statements which set forth the limitations of the value of a product, as well as to state in terms that are readily understood the quantity of vitamins or minerals present. The fact that a product may be considered a food for special dietary use does not release it from the obligation of listing ingredients by their usual or common name.

Extensive fortification of foods may possibly lead to increased and more difficult problems of control from the standpoint of assuring the consumer that the product has the vitamin content claimed. That, however, should be no deterrent if there is satisfactory evidence that an important portion of our population would benefit from such vitamin additions to staple food products. It is important that if fortification is recommended, such fortification be substantial so that the product has an identity which will definitely distinguish it from the same product without fortification. I may use tomato juice as an example. Let us assume that the vita-

min C content of tomato juice varies from 12 to 30 milligrams per 100 cc. with an average of 18 milligrams. If tomato juice were to be fortified with vitamin C—and I will interject that such fortification seems entirely unnecessary—the vitamin C content of the fortified product should be definitely higher than the vitamin C content of any tomato juice that has not been fortified.

In our control work we are conscious of an extensive, rapidly growing, and constantly changing industry in vitamin preparations. Changes are frequently brought about by new discoveries which may be accompanied by the issuance of patents. There are at present patents which relate to the synthesis or manufacture of preparations of vitamins A, B_1, C, and D suitable for incorporation in foods.

It is difficult to obtain accurate figures of the vitamin industry in this country, but I shall try to provide you with a few that may serve to give you at least a partial picture. According to available statistics more than 95 per cent of the cod liver oil used in this country is imported. Importations for 1938 totaled more than five million gallons. More than half of this oil is used for animal feeding, but an estimate that two million gallons were used for human consumption seems conservative. If this oil retailed at $1.00 per pint, its total cost to the consumer would be $16,000,000. A considerable portion of this oil is used for the manufacture of concentrates of vitamins A and D, which may be consumed as such or put into capsules, tablets, or so-called tonic preparations. These manufacturing processes increase the cost of the vitamins to the consumer. During the past year more than $30,000,000 was spent by consumers for vitamin preparations put up in gelatin capsules. Figures released by the Bureau of the Census of the Department of Commerce show that there was a five-fold increase in the manufacture of vitamin preparations made for sale directly to the public and which can be classified as U. S. Pharmacopoeia or National Formulary articles, in the two years from 1935 to 1937. During the same

period there was only a slight increase in the value of products manufactured which were intended for direct sale to or prescribed by the physician. The total value of the manufactured products for the year 1937 was in excess of $26,000,000. These figures are the value of the products as manufactured and not the cost to the consumer. To this must be added the cost of advertising, transportation, and profits of the manufacturer, the wholesale merchant, and the retailer. On the basis of the figures I have quoted and other information available to me, I have reason to believe that during the year 1938 the people of the United States spent more than $100,000,000 for vitamin preparations manufactured or sold through pharmaceutical channels. This is approximately $1.00 per person for every man, woman, and child in this country.

From the nature of the program I anticipate that the need for the actual fortification of foods will be adequately discussed this evening by other speakers. However, I would like to make a few observations. Surveys which have been made indicate that if there are deficiencies of vitamins or minerals in an important proportion of our population, such deficiencies are most likely to occur among the low-income groups. Any program of fortification will not serve its most useful purpose unless these groups are given prime consideration. Much too frequently generalizations with respect to vitamin or mineral deficiencies are found to be in error. It is a fact that, generally speaking, our foods are deficient in vitamin D. Nevertheless, since beneficial effects of vitamin D can be obtained by exposure to sunshine, it is equally true that for a large area of the United States no beneficial effects can be expected from the addition of vitamin D to foods. In spite of this fact we have seen an advertising campaign conducted for the use of vitamin D milk in Miami, Florida. It is the opinion of a number of authorities in the field of nutrition that our dietaries may be low in calcium. Yet there are extensive areas of the United States where calcium deficiency is not likely to occur. Not only should each vitamin or mineral be

considered independently, but the desirability of fortifying specific foods should also be considered independently. In trying to reach a decision with respect to the desirability of fortifying a particular food or class of foods with one of the vitamins or one of the minerals, it may be well to bear in mind that a statement justifying the fortification of a food with vitamin B_1, for example, may be used to the same advantage in promoting the sale of a vitamin B_1 preparation. I make note of this because writers have frequently emphasized the need for a certain food essential in a manner that inadvertently places them in a position of making a general recommendation. In this connection I can do no better than quote the last two sentences of a paper by H. H. Mitchell:

> "... At a time when popular periodicals are widely publishing irresponsible articles on vitamins, ignorantly, or deliberately creating an entirely distorted popular conception of them, and when commercial concerns are widely advertising purely hypothetical advantages of vitamine preparations, it is particularly important that investigators in nutrition exert great care in the wording of statements as to the practical significance of vitamines in every-day life. Otherwise they may become unwilling accomplices in the perpetration of a gigantic fraud upon the American public."

This statement was published in *Science* in July 1922, and it is in my opinion equally applicable at the present time.

RETENTION, RESTORATION, AND FORTIFICATION OF VITAMINS IN FOODSTUFFS

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I VENTURE to use a wide title. Also, to make it clear that the views to be presented are interpretation of personal experience, conviction, and policy, since the food industries have never gone on record. What follows is believed to be rational *nutritionally* and practicable *technically,* in objective and procedure.

At the outset, qualifications must be stated. The requirements of the vitamins are not accurately known for different ages and body weights in the two sexes. The vitamin contents of different foods vary with type, variety, selection, state of maturity, season, technique of harvesting, conservation, processing, and shelf-life. Thus, some of the wide variations in reported analyses are derived from original materials, others are due to differences in behavior of test animals, while still others proceed from technique of testing.

It is therefore necessary to speak of ranges of vitamin contents rather than of points. At the same time, it is natural to have a leaning toward larger intake, that is toward adequate protection; and this is apparently justified by the experience that within reasonable limits, so far as we are aware, over-dosage of vitamins is impossible in foodstuffs, perhaps with the exception of vitamin D. As the intake of vitamin rises and falls, storage within the body rises and falls, and elimination rises and falls; under these circumstances, fear of intoxication is scarcely to be entertained, except in drug-use of vitamin concentrates. This view is supported by our knowledge of metabolic actions of vitamins. Optimal intake of vitamins by a people ought to be sought by popular choice in enlarged natural intake.

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I venture to suggest that a useful division, in respect of content of vitamins in foods, may be made under headings of *retention, restoration,* and *fortification.* By *retention* is meant such technique as brings to the dining table the native content of vitamin possessed by the food. Perhaps the best illustration is vitamin C, where it has become possible to have raw fruits and vegetables and canned fruits and vegetables contain at the moment of ingestion the content of vitamin C originally possessed. With many foodstuffs, some loss in vitamins occurs in processing or cooking. With certain foods, retention is technically easy; with others difficult. Still more important, with certain foods retention of vitamins is commercially easy and in conformity with consumers' tastes and specifications; with other foods, retention is commercially difficult or not in conformity with consumers' tastes and specifications.

It is not technically easy to retain vitamin B_1 in stable milled products of grains and in raw sugar. Furthermore, the taste of the public is against whole-grain meal and raw sugar, on grounds of color and other attributes. More important is the circumstance that whole-grain meal and raw sugar are prone to decomposition, and therefore do not lend themselves to distribution in a wide country with a large population. Any one who desires whole-wheat flour or raw sugar can obtain them on the market, at a price reflecting risk of processing and distribution; but the records of milling and of sugar refining show how small is the number of persons wishing to do so. We therefore have the established fact, both here and abroad (as is shown in the public reaction against compulsory high-extraction flour in Germany and Italy), that the taste of the public demands refinement in flour and in sugar.

Refinement in flour and in sugar carries with it heavy loss of vitamin B and of minerals. Here, where retention fails, restoration, *protective restoration* if you please, ought to find application. In other words, it ought to become commercially expedient, as it is already technically practicable, to restore to white flour the vitamin

B_1 (and B_2 if desired) removed in the act of milling. It is entirely practicable to take a properly pulverized synthetic vitamin B_1 and add it to flour, intimately mixed, to restore the B_1 to the whole-grain level, which may be taken as top limit of 70 international units per ounce. It is merely a question of price; but this is at the moment entirely beyond practical application. The manufacture of synthetic B_1 has scarcely begun, and simplification of the process will doubtless result in pronounced reduction in cost. In short, *protective restoration* with synthetic vitamin B_1 is feasible in milled products technically, but at present impracticable on grounds of price. Similarly, it is practicable in the technical sense to add synthetic vitamin B_1 to ready-to-eat cereal products, such as breakfast food, crackers, biscuit, etc.

Concentrates of natural vitamin B_1 may be prepared with a minimum of color, odor, and flavor, which presumably could be used in ready-to-eat grain products; but I have not seen such preparations that could be added to flour. An indirect method of restoration of vitamin B_1 provides a new yeast, which is so rich in vitamin as to yield a bread approaching whole-wheat bread in this respect, of course at a considerable increase in cost of yeast per loaf.

In the case of vitamin B_2, the problem is somewhat different. If we attempt restoration with natural vitamin B_2 and prepare, let us say, extracts of yeast or of rice polishings, we shall secure both B_1 and B_2 in variable proportions. Personally I have never seen such extracts adapted for use with flour, or even for ready-to-serve cereal products—though this is merely a question of refinement of concentration and purification. Concentrated extracts of B_2 are to be obtained from milk-whey and also from special yeasts, again with problem of purification and concentration. Synthetic riboflavin is available but is very expensive. To what extent, however, in the protective restoration of highly milled flour, we should attempt to restore B_2 as well as B_1, is not clear from the literature.

When we come to *retention of vitamin A* and *D*, the situation is

more complex. We have here, in the first place, two kinds of retention, indirect or direct, to use the terms in a special sense. The first is potential protective retention, that is, retention of potential original content. By this I mean the higher content of vitamin A which can be given to milk (and of course to butter) and to the yolk of eggs by a diet rich in A or provitamin. We have wide variation between the vitamin A content of milk in summer and winter, if traditional methods of feeding are allowed, since most of the provitamin of grasses is lost in the usual curing of hay. It is possible (as shown in Northern Europe with the use of the Virtanen method) to have the vitamin A content of milk maintained throughout the year by appropriate feeding. Therefore, it is technically possible, wherever practicable, to maintain a high level of vitamin A content of milk through indirect retention, through diet selected to assure high A content. Direct retention of A in dairy products is also entirely practicable, in the technical sense, because there is no reason, in the proper processing of milk and butter and cheese, why vitamin A should be destroyed.

Restoration of vitamin A in milk (or butter), to top natural level, is practicable technically, though not easy because of the necessity of removal of flavor and odor of the fish-liver oil concentrate added to milk. Just as it is possible to raise the vitamin A content of milk by a diet rich in provitamin, so it is possible to add concentrated vitamin A from fish-liver oil and thus produce a protective restoration. The problem is one of price, odor, and flavor. Whether it is desirable to restore vitamin A only to butter, or also to milk, is a question which will need to be explored, though no principles of nutrition are involved.

Protective restoration of vitamin D is evidently a limited field. We are justified in the assumption that in the human being, during the summer in this latitude, there is a high rate of activation of sterols and a consequent high level of storage, which declines to a point approaching or touching deficiency during the winter. Ap-

parently this doesn't hold true in milch cows, since direct experiments with sunshine and ultra-violet light do not indicate a significant increase in formation of activated ergosterol. Apparently, under the most favorable circumstance, the natural vitamin D content of cow's milk doesn't exceed 50 international units per quart; but we are not certain to what extent this depends upon lack of ergosterol or cholesterol or inability of the ultra-violet light to penetrate. Possibly the sunlight activates secreted sterols, which are later resorbed. To feed milch cows a fortified ration is a highly specialized procedure. In any event, to undertake a restoration of vitamin D in the milk supply to the highest natural level would not be sufficient to compensate for the low incidence of ultra-violet light in winter. Here *restoration* must be supplemented by *fortification,* unless children are to rely on fish-liver oils. It is possible to feed laying hens in such a way as to produce a high vitamin D content of the yolk of the egg, but this is hardly capable of nation-wide introduction and acceptance. Also, it is possible to restore vitamin D by direct radiation of milk.

It does not seem possible to enter into any discussion of the subject of restoration of nicotinic acid and vitamin E, since so little is known of requirements or deficiency.

The diet of the majority of families, even in a country with a high standard of living (like the United States) is determined more by purchasing power than by consumers' choice or knowledge of nutrition. In an impressive manner, increase in family income is accompanied by enlarged use of protective foodstuffs and lowered use of staples. Therefore, any effort on the part of processors to improve the diet through retention and restoration, must seek to accomplish this without significant increase in cost.

Coming now to the subject of *fortification,* it seems desirable to define this as *supplementary addition, in excess of natural limits.* To add, let us say, 60 international units of synthetic vitamin B_1 to flour could be called restoration; but to add 120 would certainly

need to be called fortification. It would seem to be the natural point of view to try to be satisfied with restoration whenever possible and to proceed to fortification only under unusual or extreme circumstances. Following this view, it may fairly be stated that restoration of vitamin B_1 and B_2 in cereal products is all that need be undertaken; fortification in our country seems unnecessary. In the same sense, it may be urged that restoration of vitamin A is all that is needed, since ample intake is assured if the nation's milk supply contained the highest natural level. But in the case of vitamin D, this does not hold for all regions.

Apparently, if we are to recommend the addition of vitamin D to foodstuffs, in order not to rely on fish-liver oils, restoration is not sufficient and fortification will need to be considered. On the assumption that such fortification lies naturally in dairy products (since vitamin D is fat-soluble), this would imply the addition of vitamin D to milk. Such addition has already been accepted by the American Medical Association, applicable to milk, up to the present not applicable to butter. The amount that would be added in fortification under such circumstances would represent the amount of vitamin D known to be sufficient during the winter for the protection of children against rickets; the figure at present employed by the American Medical Association, 400 international units to the quart, is certainly adequate for this purpose. This may be added in the form of natural vitamin concentrates, as synthetic vitamin D, or may be contributed by intensive direct irradiation.

There is at present considerable cross-action, if we might so term it—that is, tendency to incorporate B_1 and B_2 into fatty foods and to add vitamins A and D to cereal foods. I cannot repress the personal conviction that this now popular tendency is temporary, and in the long-term view it will be found better to confine restoration and fortification to foods in which vitamins naturally occur. This would mean for the present, until further information is available, that vitamins B_1 and B_2 be added to cereal foods from which they

have been removed and that vitamins A and D be added to dairy products.

We must bear in mind that attempts to introduce vitamins in foodstuffs up to the optimal requirement run counter to the desire of physicians to prescribe these vitamins in cases of deficiency, and to the wish of pharmacists and grocers to sell preparations of vitamins, in capsules or tablets, for self-medication. If we review the history of the practice of medicine, we may perhaps feel driven to the expectation that in the next few years the public will stress self-medication, with vitamins purchased in concentrated form and sold in drug stores, and perhaps in groceries, on a scale in excess of use of these vitamins in restoration and fortification of foodstuffs. Whatever may be the function of students of nutrition in safeguarding the interest of the public in the practice of medicine, it ought at least to be our endeavor to safeguard the interest of the public within the food field. It is in this sense that I suggest the separation of *retention, restoration,* and *fortification,* as actors to play successive stages, in the hope that under such rational procedure the administration of vitamins may be used as much as possible in foodstuffs and least in the drug field, where inevitably self-medication will supersede prescription by physicians. We should *first* seek *retention* of native vitamins, *then restoration* when advantageous, *then fortification* when warranted, leaving medication to the last, applicable to regions and groups where ingestion of vitamins as food components encounters exceptional difficulties. In the history of the dietary use of iodine we find some confirmation of this view. If one were to adopt an ultra-modern point of view, one might perhaps regard foodstuffs merely as carriers of protein, fat, and carbohydrate, and endeavor to supply the accessories, including vitamins and minerals, more or less synthetically in tablets. Most of us, however, are not yet prepared to turn the diet over to chemists and pharmacists, or to undertake the dieting of our race as we ration experimental animals. We must separate *prevention* from *treat-*

ment of vitamin-deficiency diseases. Clear-cut clinical deficiencies usually respond best to concentrates in capsule or tablet, though hypodermic medication in heroic doses is often called for. But prevention of deficiency through adequate intake of vitamins should be sought in the food supply, in which vitamins have been retained, restored, or appropriately fortified.

At the same time, it is to be recognized that if synthetic production of vitamins proceeds to the point of very low costs, these may be lower than the costs of natural vitamins, under circumstances of prevalent methods of distribution of foodstuffs. It has become fashionable in scientific circles to predict the factory synthesis of protein, carbohydrate, and fat, in replacement of animals and crops. Certainly the synthesis of vitamins is far closer than is the synthesis of protein, carbohydrate, and fat. Eventually, therefore, it is conceivable that sometime in the future vitamins may be obtainable for mass nutrition cheaper by synthesis than from natural sources. Until that moment arrives, however, it will be wise to continue to seek and develop reliance on natural vitamins, in natural foodstuffs, appropriately modified by retention, restoration, and fortification.

FURTHER OBSERVATIONS ON THE OCCURRENCE OF RHEUMATIC MANIFESTATIONS IN THE FAMILIES OF RHEUMATIC PATIENTS[1]

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In a previous paper (1) the frequency of rheumatic manifestations was shown to be significantly higher among the near relatives of a series of children affected with rheumatic disease than among the corresponding relatives of a control series of non-rheumatic children. The findings were not conclusive, but did suggest that inherited predisposition may be an important factor in the development of the disease, and indicated the need for further investigation. The purpose of this article is to present additional facts bearing on this subject.

Material and Methods

The data upon which this report is based consist of the medical histories of ninety-six consecutive admissions of white children to the Cardiac Clinic of the Harriet Lane Home because of some rheumatic manifestation, and of thirty-three white children examined in the Tuberculosis Clinic of the Harriet Lane Home. The histories include a careful epidemiological study of the immediate families of these children, and the families of their parents, so that accurate information is available regarding the rheumatic history of the patients and their siblings, parents, grandparents, uncles, and aunts.

The children admitted to the clinics, and who are responsible for

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[2] From the Cardiac Clinic of the Harriet Lane Home (Department of Pediatrics) of the Johns Hopkins Hospital in cooperation with Child Hygiene Investigations of the United States Public Health Service and the Departments of Epidemiology and Biology, Johns Hopkins University, School of Hygiene and Public Health, Baltimore. Acknowledgment is made by the authors of the inspiration and advice of the late Dr. W. H. Frost who guided the early stages of this study.

the inclusion of their respective families in the study, are denominated "index cases" to distinguish them from their relatives who enter the study because of their relationship to these cases.

In this paper, as in the preceding one, a rheumatic manifestation is defined as either chorea, rheumatic fever, or rheumatic carditis. All index cases of the rheumatic group entered the clinic because they were suffering from one or more of these conditions. The index cases who were children examined in the Tuberculosis Clinic, and their relatives, are used as a control group and none of the index cases in this group has had an acute rheumatic episode.

The selection of the material, verification of the information, and comparability of the two groups were discussed fully in the previous article, which dealt with a smaller group of rheumatic families than is here analyzed. It should be emphasized again that the two groups of families are comparable for such factors as age, mortality, and social and economic status.

THE ANCESTRY OF RHEUMATIC AND CONTROL CHILDREN

A comparison of the rheumatic and control index cases, with respect to the history of rheumatic manifestations in their parents and grandparents, is presented in detail in Table 1. This table shows the various parental and grandparental mating combinations with respect to the occurrence of rheumatic disease, and the number of index cases in both the rheumatic and control series for each combination. This table may be summarized as follows:

Number of Index Cases		*Rheumatic*	*Control*
With Parental Histories Complete		96	33
With Rheumatic History in Parents	Number	43	4
	Per cent	44.8	12.1
With Grandparental Histories Complete		86	32
With Rheumatic History in Grandparents	Number	49	4
	Per cent	57.0	12.5

With Complete Parental and Grandparental Histories		86	32
With Rheumatic History in Parents *or* Grandparents	Number	63	7
	Per cent	73.3	21.9

It was possible to obtain complete histories with respect to the occurrence of rheumatic manifestations on every parent of all the rheumatic and control index cases, but the history with respect to rheumatic disease is complete in the grandparents of only eighty-six

Table 1. Distribution of index cases of rheumatic and control families according to the correlation of the history of rheumatic manifestations in the parents and grandparents.[1]

Grandparental History				Rheumatic Index Cases					Control Index Cases				
Paternal		Maternal		Parental History					Parental History				
GF	GM	GF	GM	F M − −	F M + −	F M − +	F M + +	Total	F M − −	F M + −	F M − +	F M + +	Total
−	−	−	−	23	3	9	2	37	25	3			28
−	−	+	−	9	1		2	12					
−	−	−	+	3	2	6		11	2				2
−	−	+	+	2		2		4					
+	−	−	−	5	1	1	2	9					
+	−	+	−										
+	−	−	+			1		1	1				1
+	−	+	+										
−	+	−	−	4	1	2	1	8	1				1
−	+	+	−	1				1					
−	+	−	+		1	1		2					
−	+	+	+										
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+	+	−	+	1				1					
+	+	+	+										
?	−	−	−	1				1					
?	−	−	+			2		2					
?	−	+	+			1		1					
?	−	?	−	1				1					
?	?	−	−	1				1			1		1
?	?	−	+			2		2					
−	−	?	?	2				2					
Total				53	9	27	7	96	29	3	1		33

[1] F = father. M = mother. GF = grandfather. GM = grandmother.

of the rheumatic and thirty-two of the control index cases. The discussion, therefore, of the proportion of index cases with history of rheumatic manifestations in their grandparents is limited to those cases for whom the information was complete.

Of the ninety-six rheumatic index cases, forty-three, or 44.8 per cent, had one or both parents with a history of rheumatic disease in the past as compared with four, or 12.1 per cent, of the thirty-three control index cases. Thus the percentage of index cases with rheumatic parents in the rheumatic group was 3.7 times as high as that of the control group. The percentage of index cases who had grandparents with a positive history in the rheumatic group was almost five times that found in the control group, the percentages being 57.0 to 12.5.

When both parental and grandparental histories are considered, the percentage of index cases with at least one parent or grandparent giving a history of rheumatic manifestations was 73.3 per cent in the rheumatic group as compared with 21.9 per cent in the control group. These results show quite definitely that a much greater proportion of the rheumatic index cases have parents or grandparents who have had rheumatic disease than is found in the corresponding relatives of the control index cases. They demonstrate in another form the findings of the previous article, which showed that there was an unusual occurrence of this disease in the families of rheumatic index cases.

The findings with respect to the percentage of rheumatics who have parents with rheumatic histories is, moreover, consistent in the two generations analyzed. Forty-three of the ninety-six rheumatic index cases had parents with a history of rheumatic disease. A study of Table 1 shows that thirty-six of these had one parent rheumatic and seven had both parents rheumatic, or there were in all fifty rheumatic parents. A summary of the history of rheumatic manifestations in *their* parents (grandparents of the index cases) compiled from Table 1 shows for these rheumatic parents:

Number of Rheumatic Parents of Index Cases		50
With Rheumatic History in *Their* Parents	Number	23
	Per cent	46.0

This percentage is in agreement with the percentage of index cases whose parents gave a history of rheumatic manifestations seen in the summary of Table 1, and shows the consistency of this finding in two generations of these rheumatic families.

THE OFFSPRING OF RHEUMATIC AND NON-RHEUMATIC PARENTS

The families in this study were selected in two ways, (1), those of the rheumatic index cases because at least one child, the index case, came to the clinic with some form of rheumatic manifestation, and (2), those of the control group because the index case was examined in the Tuberculosis Clinic and had not had an acute rheumatic episode. Thus, by definition, at least one child in each of the rheumatic families must be rheumatic, and one child in each of the control families must be non-rheumatic.[8] Because of this method of selection, the immediate families of the index cases are not suitable for a direct comparison of the relative frequency of rheumatic infection in the offspring of the parental matings.

The information obtained for the grandparents and *their* children may, however, be used for this purpose, because selection of the families was entirely independent of any prior knowledge of the past history with respect to rheumatic manifestations of the grandparents, parents, uncles, or aunts of the index cases. The findings of this analysis are, therefore, not comparable with the findings of other investigators who used the immediate families of their index cases to study this relation (2, 3, 4).

These grandparental families also have the advantage in that they are complete; the grandparents are all past the reproductive

[8] It should be noted that control families were selected without reference to the past history of rheumatic manifestations in the siblings of the index case so that it could happen that some of the siblings had been registered in the Cardiac Clinic—this did occur in three instances.

age and their living offspring are mostly past the age of maximum incidence of rheumatic disease. It is also permissible to combine data on both the rheumatic and control families, because in this generation any difference which might be shown between the two groups is not due to the method of sampling but should rather be considered due to the selectivity of the disease.

The total number of grandparental families in the combined rheumatic and control groups was 258, and complete information was available with respect to the history of rheumatic manifestations in the parents and children of 246 of these families (see Table 1). From the 246 matings, for which complete histories are available, there are 1,303 offspring, of which 150 had a history of some type of rheumatic manifestation.

The distribution of the grandparents of index cases according to their history of rheumatic disease, the number of offspring, and the number and percentage of rheumatic offspring from each type of mating is shown, by sex, in Table 2.

A study of this table shows that where one or both parents

Table 2. Distribution of grandparental families of index cases according to the history of rheumatic disease in the parents, with the number of offspring of each type of mating, and the number and percentage of rheumatic offspring, by sex.

	History of Rheumatic Manifestations in Parents of Grandparental Families (Grandparents of Index Cases)					
	Male − Female −	Male + Female −	Male − Female +	Male + Female +	Unknown	Total
Number of Matings	182	24	34	6	12	258
Male Offspring						
Number	491	61	85	18		655
Number Rheumatic	31	8	16	6		61
Percentage Rheumatic	6.3	13.1	18.8	33.3		
Female Offspring						
Number	483	40	104	21		648
Number Rheumatic	42	7	34	6		89
Percentage Rheumatic	8.7	17.5	32.7	28.6		

(grandparents of index cases) gave a rheumatic history a much higher percentage of the offspring (parents, uncles, and aunts of index cases) were affected than when neither mate had manifestations. There are, in addition, some interesting sex differences which are worthy of note. From the thirty-four matings of a rheumatic female with a non-rheumatic male there were 104 female offspring of whom thirty-four were affected, and eighty-five male offspring of whom sixteen had manifestations. Rheumatic mothers thus had almost twice the percentage of rheumatic children among their female offspring than they had among their male offspring. This is in contrast to the fact that no difference is noted in the percentage of male and female offspring who were rheumatic in the twenty-four families where the father was rheumatic and the mother non-rheumatic. Viewed from another angle, we note with respect to the male offspring, that there was apparently no difference in the percentage who were rheumatic in the families in which the mother gave a positive history and those in which the father gave a positive history. Such was not the case with the female offspring, the percentage of female offspring who were rheumatic being almost twice as high in the families where the mother was rheumatic as it was in the families of rheumatic fathers.

The higher percentage of rheumatic children found among the offspring of rheumatic parents than among the offspring of non-rheumatic parents suggests that the hereditary constitution may be a factor in determining predisposition to this disease. The higher frequency of rheumatic disease among female offspring of rheumatic mothers would seem to indicate that if heredity is a factor a sex difference exists.

RHEUMATIC MANIFESTATIONS ON THE PATERNAL AND MATERNAL SIDES OF FAMILIES OF RHEUMATIC INDEX CASES

A comparison of the percentage of the offspring of the paternal and maternal grandparents of the rheumatic index cases who gave

a history of rheumatic manifestations yields further information upon possible hereditary relationships. In making such a comparison, the parents of the index cases must be excluded, because by the method of selection of these families, there must be one male child in each family of paternal grandparents and one female child in each family of maternal grandparents. Any hereditary factor present in these families would also manifest itself most strongly in the parents of the rheumatic index cases since if such a factor be present, they must, of necessity, be the transmitters of the disease.

Another consideration is the accuracy of the histories obtained, because it is generally much easier to interview the mother than the father of the index case, and for this reason only those families on the paternal side were considered in which the history of rheumatic disease was obtained from the father or one of his sisters.

The histories of eighty-six paternal and ninety-six maternal families are believed to be accurate, and these have been analyzed to show the percentage of aunts and uncles (siblings of parents of index case) with a rheumatic history on the paternal as compared with the maternal side. This comparison is shown in Table 3.

A study of this table reveals the interesting fact that while there is no difference between the proportion of the uncles and aunts who have rheumatic manifestations on their respective sides of the family, the percentage of maternal aunts and uncles who had rheumatic manifestations is almost three times that of the paternal aunts and uncles.

Table 3. Comparison of the relative frequency of rheumatic manifestations in the aunts and uncles of rheumatic index cases on the paternal and maternal sides.

Relation to Index Case	Paternal			Maternal		
	Number	Rheumatic		Number	Rheumatic	
		Number	Per Cent		Number	Per Cent
Uncles	159	4	2.5	213	33	15.5
Aunts	172	9	5.2	225	35	15.6

As these aunts and uncles enter the study because they are siblings of the parents of the index cases, any interpretation of the above finding is dependent to some extent upon whether the difference noted is due to the fact that in this group of families a greater number of mothers than fathers of index cases had a history of having had rheumatic disease (see Table 1). A further analysis has therefore been made in which the aunts and uncles are classified as siblings of rheumatic and non-rheumatic parents to compare paternal and maternal sides of these families according to the history of the parent through whom they are related to the rheumatic index case. This comparison is shown in Table 4.

This table amplifies the findings shown in Table 3 and demonstrates the fact that, when due consideration is given to the presence or absence of a history of rheumatic disease in the parents of index cases, a greater proportion of the maternal than of the paternal aunts and uncles is found to be affected. The number of persons falling into each group in this table is not sufficiently large to allow significance to be attached to the individual percentages, but they are consistent in that, in each instance, they show a higher per-

Table 4. Comparison of the relative frequency of rheumatic manifestations in the aunts and uncles of rheumatic index cases on the paternal and maternal sides; according to the history of rheumatic manifestations in the parents of whom they are siblings.

Sibling of	Relation to Index Case	Paternal			Maternal		
		Number	Rheumatic		Number	Rheumatic	
			Number	Per Cent		Number	Per Cent
Rheumatic Parents of Index Cases	Uncles	23	1	4.3	105	21	20.0
	Aunts	26	3	11.5	95	25	26.3
	Total	49	4	8.0	200	46	23.0
Non-rheumatic Parents of Index Cases	Uncles	136	3	2.2	108	12	11.1
	Aunts	146	6	4.1	130	10	7.7
	Total	282	9	3.2	238	22	9.2

centage of persons with rheumatic disease among the maternal aunts and uncles than among the corresponding relatives on the paternal side of the family.

This supplements the observation, brought out by Table 3, that more rheumatic disease occurred on the maternal than on the paternal side of these families of rheumatic index cases, and indicates that this difference is present whether the parents be rheumatic or non-rheumatic. This fact is worthy of consideration in any attempt to evaluate the hereditary factors in rheumatic disease.

Summary and Conclusions

Facts have been presented relative to the high incidence of rheumatic disease in the families of ninety-six rheumatic children. The percentage of persons with a rheumatic history, who had parents with a similar history, was found to be consistent in two generations of these families and was 3.7 times as high as was found in a group of control families.

The offspring of the grandparents of the rheumatic and control index cases were studied to see if any relationship was present between the type of mating with respect to rheumatic disease and the percentage of children who were rheumatic. When one or both parents had a history of rheumatic manifestations a greater percentage of the offspring was rheumatic than was found in the offspring of parents who gave no history of rheumatic disease.

The percentage of female offspring of rheumatic mothers who had rheumatic manifestations was found to be almost twice as high as in the male offspring of these mothers.

A greater percentage of persons with rheumatic disease was found among the maternal aunts and uncles than was found among the paternal aunts and uncles of rheumatic index cases.

These findings suggest that hereditary constitution may play a rôle in the predisposition to this disease. The evidence here presented does not, however, exclude the possibility that infection

plays an important rôle, and that exposure may be the predominating factor.

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THE EFFECT OF TUBERCULOSIS ON THE SIZE OF FAMILY[1]

JEAN DOWNES

VARIOUS studies have shown that there is a greater risk of disease and death among persons in intimate familial contact with infectious tuberculosis than is true of persons in the general population.[2] However, the full effect of tuberculosis upon the growth of the family as a simple biological unit has not been described in precise terms. Excluding collateral or subsidiary relationships, the family as a simple biological unit may be considered as composed of the husband, the wife, and their offspring. The size of the simple family unit at any given moment of time after marriage is determined by the operation over a period of years of two factors: namely, the number of births which determines the increment of the family, and the number of deaths which occur among both offspring and parents. It is of considerable interest to observe the effect of the operation of these two factors upon families where one or both parents had tuberculosis contrasted with the experience of families drawn from the general population.

[1] From the Milbank Memorial Fund.

[2] Weinberg, W.: Die Kinder der Tuberkulösen. Leipzig, Verlag von S. Hirzel, 1913.

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To observe the effect of the birth rates and the death rates upon the size of the natural family unit, it is necessary that families be followed over a considerable period of years. Births may occur over a period of twenty-five to thirty years of married life, and disease and death may occur at any time during the life of the family. Through special investigation and study of family histories in Cattaraugus County, New York, it is possible to show the growth of the natural family as a unit, as evidenced by (1) a group of tuberculous families in which marriage occurred and the children were born during the latter half of the nineteenth century, and (2) a group of families in a general population drawn from the same area and period of time. It is possible, also, to study similar groups of families in the present century and to contrast their experience for a limited period of time after establishment of the family with those of the past century.

DATA AND METHOD OF STUDY

In connection with a study of morbidity conducted in a rural area of Cattaraugus County by the United States Public Health Service in cooperation with the Milbank Memorial Fund, an effort was made to secure a history of the parents and sibs of both the husband and wife in each of the 1,400 families in the morbidity survey. The data included in the history which are pertinent to this particular study are: (1) the date of marriage and (2) the date of birth, (3) the age and date of death for each member of the family and (4) the cause of death for all who were deceased at the time the survey was made.* Every effort was made to secure as complete and accurate family history records as possible. In many instances the family Bible was used as a source of information, or at least as a check of the accuracy of the data obtained from the informant. When there was uncertainty as to the date of death or age at death for a deceased member of the family and the place of burial was known, the ceme-

*The family histories were secured during the period 1931-1933. The family, as considered in this study, is the simple family unit consisting of a husband and wife and their offspring.

tery was visited and this information was taken from the gravestone. If the grave could not be located, the records of the sexton of the burying ground were consulted for the needed information.[4] Occasionally, a part of the family history record was obtained through questionnaires sent to surviving members of the family or relatives living in various parts of the United States. In fact, all available channels were used in an effort to secure a complete record for each family.

In a rural community, such as the one from which these records were obtained, the ages of the husbands and wives in the present families will vary from those in their early twenties or younger to the very old, that is, those who have survived to past eighty years of age. Consequently, such records will include some families in which the offspring were born previous to 1850 as well as some in which the offspring were born since 1900. The data for this particular study consist of records for 892 families in which all the children were born previous to 1901.[5] The majority of these families had lived in Cattaraugus County or were from near-by rural areas. For the purposes of this study they may be considered as a sample of rural families in which the offspring were born in the latter half of the nineteenth century.

In calculating the fertility experience of the 892 married pairs, the exposure to risk is controlled by taking the length of marriage for each pair into account. When the death of either the husband or the wife occurred, the couple was dropped from the population.

[4] Records of all burials in a given cemetery are kept by the sexton. In many instances these records go as far back as the middle of the eighteenth century. The record for each deceased person includes the name of the father and the mother of the deceased, the date of death and the cause of death of the deceased. The investigators were always permitted to examine the sexton's records.

[5] When data concerning date of marriage were lacking, or date of birth or age at death or cause of death were lacking for some members of the family, the entire record was discarded; also records for foreign-born families were excluded. In all, 10 per cent of the total family histories were considered unsuitable for use because the date of birth or the age at death of the wife or husband and the date of marriage were lacking. Also, 16 per cent of the family histories had to be discarded because data in regard to the offspring were lacking or the families were foreign-born.

In calculating the size of family, each year of life or fractional year of life of each person in the family after the date of its establishment, that is, the date of marriage, is considered, and the total person-years derived in this manner constitute a total population in each given year of observation of the family. This total population in any given year of observation, divided by the total number of families considered, indicates the average size of the family unit.

Both the fertility and the mortality experience of a group of married pairs, derived from family history records secured in the manner described above, have an obvious bias. The childless family will not be represented, for the securing of the family history was dependent upon an offspring. The mortality experience of a population secured from family history records is also weighted because a survivor in the family was a requisite for obtaining the record in practically all instances.[6] Families living in the vicinity in the same time-period but which have died out completely are necessarily not included. Furthermore, the probability of having a surviving offspring in the community is influenced by the original number of offspring in the family. However, it is believed that these limitations of the data in respect to fertility and to mortality do not preclude their use for purposes of internal comparison since the biases pointed out above are common to all the families. But it should be emphasized that the factor of survivorship as an essential in securing the family records operates most forcibly upon the tuberculous families, where due to excessively high mortality the chance of survival of offspring was considerably less than was true of those in the general population.

FERTILITY IN TUBERCULOUS FAMILIES IN THE PAST AND PRESENT CENTURIES

Included in the sample of 892 families, or married pairs, are thirty-eight in which it is recorded that either the husband or wife

[6] In a few cases where the wife and all members of her family were dead, the wife's family history was secured from the husband, and vice versa.

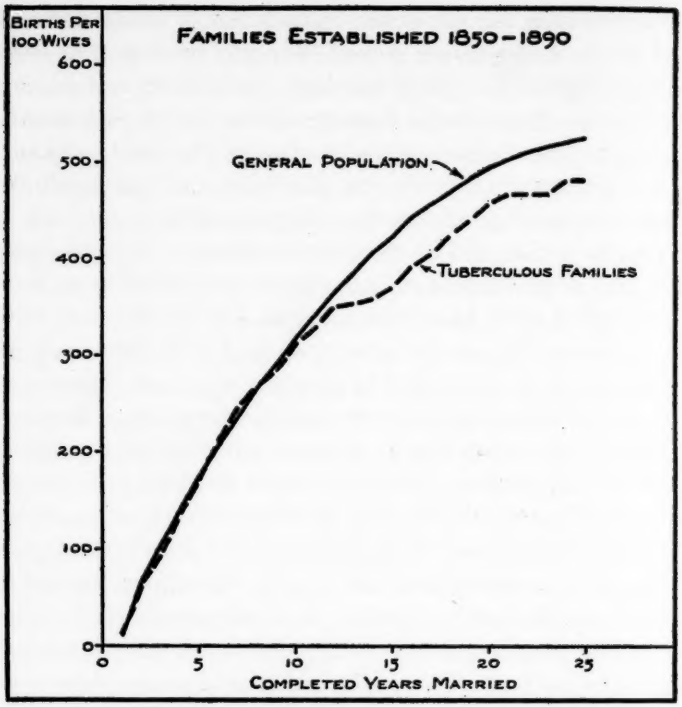

Fig. 1. Cumulated birth rates for successive years of married life among two groups of women in the nineteenth century.

or both died of tuberculosis before reaching the age of fifty years.[7] Figure 1 and Table 1 show the cumulated birth rates through twenty-five years of married life for the tuberculous pairs compared with the birth rates for the remaining 854 pairs. The cumulated birth rates are of especial interest, for they indicate the rapidity with which the "natural family" was built up among pairs where one or both parents had tuberculosis contrasted with the average "na-

[7] The family history records do not include information concerning the probable date of onset of tuberculosis in either spouse. It was considered best to limit the group to cases where death from tuberculosis occurred prior to age fifty, which brings the occurrence of disease and death closer to the childbearing period.

Years Married	Tuberculous			General Population			Cumulative Rates	
	No. of Wives	No. of Births	Rate per 100 Wives	No. of Wives	No. of Births	Rate per 100 Wives	Tuber-culous	General Population
1	38	4	10.5	854	106	12.4	10.5	12.4
2	38	20	52.6	854	460	53.9	63.1	66.3
3	36	11	30.6	847	311	36.7	93.7	103.0
4	35	15	42.8	843	307	36.4	136.5	139.4
5	34	12	35.3	838	296	35.3	171.8	174.7
6	33	13	39.4	835	272	32.6	211.2	207.3
7	32	11	34.4	834	244	29.3	245.6	236.6
8	29	7	24.1	825	243	29.4	269.7	266.0
9	27	4	14.8	816	196	24.0	284.5	290.0
10	26	7	26.9	804	222	27.6	311.4	317.6
11	25	4	16.0	796	188	23.6	327.4	341.2
12	22	5	22.7	787	187	23.8	350.1	365.0
13	22	1	4.5	780	160	20.5	354.6	385.5
14	20	1	5.0	777	165	21.2	359.6	406.7
15	19	2	10.5	775	130	16.8	370.1	423.5
16	18	4	22.2	769	139	18.1	392.3	441.6
17	17	2	11.8	759	122	16.1	404.1	457.7
18	15	3	20.0	750	88	11.7	424.1	469.4
19	13	2	15.4	743	105	14.1	439.5	483.5
20	12	2	16.7	730	84	11.5	456.2	495.0
21	9	1	11.1	725	69	9.5	467.3	504.5
22	9	0	0	717	41	5.7	467.3	510.2
23	8	0	0	712	48	6.7	467.3	516.9
24	7	1	14.3	701	38	5.4	481.6	522.3
25	7	0	0	688	25	3.6	481.6	525.9

[1] Date of marriage 1850–1890.

Note: Every wife or couple is counted for each specific year of married life, that is, every wife who completed two years of married life is counted for that year, every wife who completed three years of married life for that year, etc., with the result that the number of wives for whom rates were computed change year by year.

Table 1. Birth rates in successive years of married life among two groups of women in the nineteenth century.[1]

tural family."[8] During the first ten years of married life the birth

[8] Since age of the wife at marriage is an important factor in considering birth rates over a period of married life, the two groups of pairs were tested for comparability in this respect. The percentage distribution according to age of wife at marriage was as follows:

Age of Wife at Marriage	*Spouse Died of Tuberculosis*	854 *Pairs*
All Ages	100.0	100.0
Under 20 Years	39.5	43.4
20-24 Years	39.4	38.5
25-29 Years	15.8	12.8
30+ Years	5.3	5.3

rates among the two groups of married pairs are strikingly similar. From that point on, births became less frequent in the tuberculous group, and throughout the remaining fifteen years of married life the cumulated rates are somewhat lower in this group than among the general population. At the end of twenty-five years of married life 100 women in tuberculous families had borne on an average 481 children compared with an average of 526 children per 100 women in the general group of families.

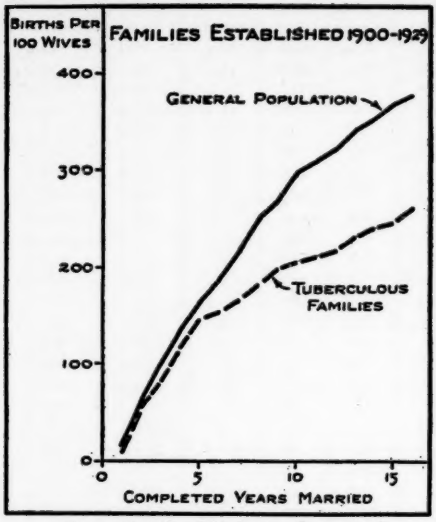

Fig. 2. Cumulated birth rates for successive years of married life among two groups of women in the twentieth century.

It is of considerable interest to examine the cumulative birth rates for a more recent period, 1900-1929, among somewhat similar groups of pairs drawn from the same area, Cattaraugus County. Figure 2 and Table 2 show the cumulated birth rates during the first sixteen years of married life for fifty-eight[9] pairs where one or the other spouse had tuberculosis, compared with the birth rates for a selected sample of 100 pairs drawn from the same rural area.[10] During the first five years of married life the birth rates

[9] Detailed records were collected and reported upon for eighty-three families in which the index case was one of pulmonary tuberculosis. In forty-eight of the eighty-three families, a parent had pulmonary tuberculosis. The families were selected on the basis of the presence of an active case in some member of the family during the period 1923-1930. An additional ten families in which a parent had pulmonary tuberculosis were obtained from records secured for all new active cases reported in Cattaraugus County during the period January, 1932 to July 1, 1935.

[10] The sample consisting of 100 married pairs was drawn from a group of some 1,400

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Years Married	Tuberculous			General Population			Cumulative Rates	
	No. of Wives	No. of Births	Rate per 100 Wives	No. of Wives	No. of Births	Rate per 100 Wives	Tuber-culous	General Population
1	58	5	8.6	100	14	14.0	8.6	14.0
2	58	27	46.6	100	48	48.0	55.2	62.0
3	57	15	26.3	100	37	37.0	81.5	99.0
4	57	21	36.8	99	34	34.3	118.3	133.0
5	52	14	26.9	96	30	31.2	145.2	164.5
6	48	4	8.3	95	22	23.2	153.5	187.7
7	47	6	12.8	87	26	29.9	166.3	217.6
8	44	7	15.9	81	27	33.3	182.2	250.9
9	41	7	17.1	79	13	16.4	199.3	267.3
10	40	3	7.5	72	22	30.6	206.8	297.9
11	34	2	5.9	71	8	11.3	212.7	309.2
12	33	2	6.1	64	8	12.5	218.8	321.7
13	30	4	13.3	62	12	19.4	232.1	341.1
14	24	2	8.3	60	6	10.0	240.4	351.1
15	19	1	5.3	57	9	15.8	245.7	366.9
16	19	3	15.8	55	5	9.1	261.5	376.0

[1] Date of marriage 1900–1929.

Note: Every wife or couple is counted for each specific year of married life, that is, every wife who completed two years of married life is counted for that year, every wife who completed three years of married life for that year, etc., with the result that the number of wives for whom rates were computed change year by year.

Table 2. Birth rates in successive years of married life among two groups of women in the twentieth century.[1]

families in five rural townships in Cattaraugus County. These families formed the population included in a special study of morbidity conducted during the period 1929-1932 by the United States Public Health Service in cooperation with the Milbank Memorial Fund. The sample of 100 was selected in the following manner: the 1,400 families were arranged according to an alphabetical file; those falling under each of the following letters—A,E,F,G, J,L,N,P,T and U—were sorted according to the date of marriage; only those with one marriage, and that occurring during the period 1900-1929, were used. These records, still in alphabetical groups, were then arranged according to age of the wife at marriage. The sample was then selected so as to be comparable with the distribution according to the age of wife at marriage among the fifty-eight pairs where either the husband or wife, or both, had tuberculosis. The percentage distribution for both groups is shown in the following table:

Age of Wife at Marriage	*Tuberculous Spouse*	*Sample—100 Pairs*
All Ages	100.0	100.0
Under 20 Years	43.1	43.0
20-24 Years	31.0	31.0
25-29 Years	15.6	16.0
30+ Years	10.3	10.0

No effort was made to have the two groups of families comparable with respect to the frequency of no-child families. In fact, the proportion of childless marriages was similar in both groups; 14 per cent of the 100 married pairs had no offspring, and 13.8 per cent of the fifty-eight tuberculous pairs had no offspring.

for the two groups of married pairs were similar. However, from the sixth to the sixteenth year of married life the fifty-eight pairs (tuberculous) had a markedly lower birth rate than was noted among the 100 pairs during the same period of married life. For example, after sixteen years of married life 100 women in the tuberculous families had borne on the average 261 children compared with an average of 375 children for the 100 women drawn from a general population.

These groups of married pairs drawn from the modern period are not strictly comparable with the groups for the previous century (Figure 1) in that those in the modern period contain childless families, and the groups in the other period of necessity do not.[11] Therefore, it is not suitable to compare the level of the birth rates in the two periods of time. However, considerable significance may be attached to the fact that the differential fertility rates in the two groups persist in the same direction both in the nineteenth century and in the twentieth century. These data suggest that when tuberculosis is present in a parent the natural family is built up with less rapidity and is on the whole smaller in size than is ordinarily true of families in the rest of the community.

SIZE OF FAMILY IN THE PAST AND PRESENT CENTURIES

The excess mortality among offspring of tuberculous parents over the average mortality in the community has been presented in a previous publication.[12] However, it is of interest to see the full effect of the operation of the two factors—the differential fertility and the differential mortality—upon the growth of the family. This may be most strikingly presented by showing the average size of the natural family, including parents and their offspring, over a period of years for the two groups of families, the tuberculous group and

[11] Fourteen per cent of the 100 married pairs had no offspring, and 13.8 per cent of the fifty-eight tuberculous pairs had no offspring.

[12] Downes, Jean: The Risk of Mortality among Offspring of Tuberculous Parents in a Rural Area in the Nineteenth Century. *The American Journal of Hygiene*, November, 1937, xxvi, No. 3.

Table 3. Size of family in successive years after establishment among the tuberculous, and in a general population in the nineteenth century.[1]

Years Family Observed	Average Size of Family		Number of Years of Life in Families		Number of Families	
	General Population	Tuberculous Families	General Population	Tuberculous Families	General Population	Tuberculous Families
1	2.0	2.0	1,746	76	854	38
2	2.6	2.5	2,188	96	854	38
3	2.9	2.8	2,484	106	854	38
4	3.2	3.1	2,760	117	854	38
5	3.5	3.3	3,024	126	854	38
6	3.8	3.6	3,283	138	854	38
7	4.1	3.8	3,502	143	854	38
8	4.4	3.8	3,716	146	854	38
9	4.6	4.0	3,886	151	854	38
10	4.8	4.1	4,073	154	854	38
11	5.0	4.1	4,237	155	854	38
12	5.1	4.2	4,383	158	854	38
13	5.3	4.2	4,511	159	854	38
14	5.4	4.2	4,632	159	854	38
15	5.6	4.2	4,758	159	854	38
16	5.7	4.2	4,870	161	854	38
17	5.8	4.3	4,955	162	854	38
18	5.9	4.3	5,029	163	854	38
19	6.0	4.2	5,099	161	854	38
20	6.0	4.3	5,152	162	854	38
21	6.1	4.2	5,208	158	854	38
22	6.1	4.1	5,224	157	854	38
23	6.1	4.2	5,242	154	854	37
24	6.2	4.1	5,255	150	854	37
25	6.1	4.0	5,249	149	854	37
26	6.1	4.0	5,239	147	854	37
27	6.1	3.9	5,232	143	854	37
28	6.1	3.8	5,215	139	854	37
29	6.1	3.6	5,198	135	854	37
30	6.0	3.6	5,153	134	854	37
31	6.0	3.5	5,146	131	854	37
32	6.0	3.5	5,109	128	854	37
33	5.9	3.4	5,079	126	854	37
34	5.9	3.4	5,047	125	854	37
35	5.9	3.4	5,016	124	854	37
36	5.8	3.3	4,978	123	852	37
37	5.8	3.2	4,938	120	851	37
38	5.8	3.2	4,895	116	847	36
39	5.8	3.2	4,830	113	840	35
40	5.7	3.2	4,772	108	839	34
41	5.7	3.2	4,750	104	833	33
42	5.7	3.2	4,735	98	826	31
43	5.6	3.1	4,569	95	816	31
44	5.6	3.1	4,471	95	803	31
45	5.5	3.1	4,356	93	787	30

[1] Families established 1850–1890.

the general group, both in the nineteenth century and during the early part of the present century. These data are presented in Tables 3 and 4 and in Figures 3 and 4. Figure 3 shows the average size of family for each year during a period of forty-five years after establishment of the family for each of the two groups of families (tuberculous and general groups) in the nineteenth century. In the general group (854 families), the size of the family increased rapidly until the fifteenth year after its establishment; at the end of that period there were on an average 5.6 persons per family. From the fifteenth to the nineteenth year the increment to the family was considerably slower; in the twentieth year the average size of family was six persons, and this level was maintained for some ten years. After thirty years of observation, as might be expected, the size of the family declined slowly and at the end of forty-five years of observation reached an average of 5.5 persons per family.

Table 4. Size of family in successive years after establishment among the tuberculous and in a general population in the twentieth century.[1]

Years Family Observed	Average Size of Family		Years of Life in Families		Number of Families	
	General Population	Tuberculous Families	General Population	Tuberculous Families	General Population	Tuberculous Families
1	2.1	2.0	205.75	117.25	100	58
2	2.5	2.4	250.00	142.00	100	58
3	2.9	2.7	286.50	156.00	100	58
4	3.1	3.0	312.25	176.00	100	58
5	3.4	3.2	340.25	184.00	99	58
6	3.7	3.2	354.50	179.75	96	56
7	3.9	3.3	357.75	180.50	91	55
8	4.3	3.3	361.50	180.50	84	55
9	4.6	3.4	364.25	175.50	80	51
10	4.6	3.5	348.25	176.00	76	51
11	4.9	3.6	349.75	163.00	72	45
12	4.8	3.6	337.50	159.50	70	44
13	5.0	3.6	325.00	151.25	65	42
14	5.1	3.7	324.50	143.50	64	39
15	5.2	3.6	328.75	121.50	63	34
16	5.3	3.8	315.50	118.00	60	31

[1] Families established 1900–1929.

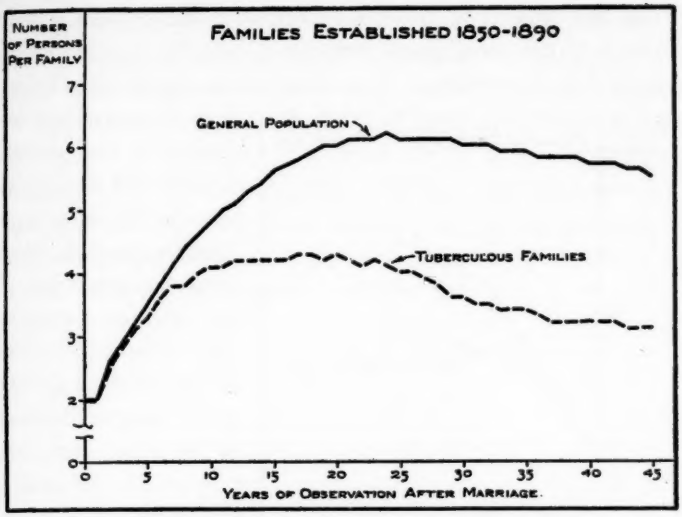

Fig. 3. Average size of family in successive years after establishment in the nineteenth century.

The curve describing the growth of the family where tuberculosis occurred in a parent differs markedly from that for the general group of families. After the tenth year of observation the tuberculous family was on the average significantly smaller in size than was true for the general group of families in the same time-period; for example, in the fifteenth year after marriage the average size of the tuberculous family was approximately four persons per family contrasted with 5.6 persons per family in the general group. Furthermore, in the tuberculous families the decline in the size of family started twenty years earlier and was much more precipitate than was noted for the families in the general group. Without doubt the natural family unit in the tuberculous group tended to be eliminated much more rapidly than was true of family units in the general population.[18]

[18] It should be pointed out that in this study persons are withdrawn from the family unit

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The two groups of families, tuberculous and general groups, drawn from the twentieth century, presented in Table 4 and Figure 4, show that the growth of the family unit during the first sixteen years after marriage was similar to the growth experienced during the same period after marriage by families drawn from the latter part of the previous century. The differential in the size of family in the two groups was similar both in the nineteenth century and in the twentieth century. For example, fifteen years after establishment of the family there was on the average in each period an excess of 1.4 persons per family in families drawn from the general population contrasted with the tuberculous families. These differences in average size of family in both time-periods (nineteenth century and twentieth century) may certainly be considered as significant. Furthermore, these data suggest that in this rural area family units in the present generation are following somewhat the same general pattern of growth as experienced by those in the past generation.

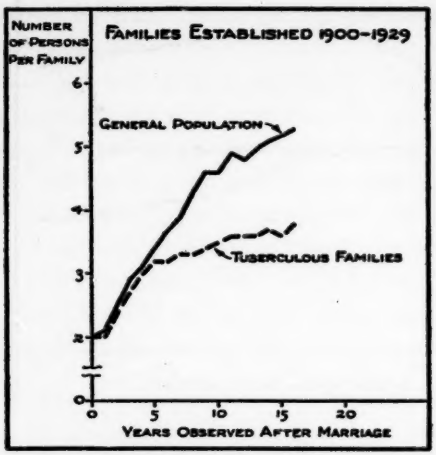

Fig. 4. Average size of family in successive years after establishment in the twentieth century.

Even though the data in this study are based upon small samples drawn from a rural area it seems suitable to call attention to their wider meaning. The tendency of the tuberculous family unit to be eliminated more rapidly through a somewhat lower fertility operat-

only because of death. This applies both to parents and to offspring. Also, offspring are counted as a part of the biological unit throughout their lives up to the date the family record was secured, even though they may have left the household.

ing in conjunction with an excessively high mortality among offspring has probably been a factor contributing to the decline in the tuberculosis death rate which has been occurring over a long period of years. Limitation of births among the tuberculous is now being encouraged as a therapeutic measure, and if in the future there is no marked change in the hazard of disease and death to the offspring of the tuberculous, we may expect the size of the family unit among the tuberculous to play an increasingly important part in the continued decline in mortality from the disease.

DIFFERENTIAL REPRODUCTION IN ENGLAND

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PRESENT-DAY differences in reproduction between social-economic groups are usually studied in terms of legitimate fertility rates standardized for age of married women. This procedure is often apt to yield misleading results because it does not take into account three variables: marriage frequency, marriage age, and extra-nuptial fertility. Among these marriage age is the most important. Women in the United States whose husbands are engaged in professional occupations marry about four years later than do women in the unskilled labor class. This alone makes the legitimate fertility rate of the late marrying group appear higher by about one-fifth. This source of error may be avoided by standardizing not for age of wife but for duration of marriage. If this is unfeasible because the duration of marriage is unknown, then it seems advisable to group births by age of father rather than of mother and to relate them not to the number of married men in the corresponding age groups, but to the total number of men married and unmarried combined. The age-specific "paternity rates" may be computed for single years of age or else on a quinquennial or decennial basis as the material may permit. Their summation over the whole reproductive life span yields a "total paternity rate" which gives the number of births per 1,000 men during their lifetime.

If only the total number of births in each social-economic group is known, it is still possible to estimate the total paternity rate by substituting standard rates for each age group and adjusting them according to the ratio of observed to expected births. Since in all western population groups about 50 per cent of the fathers are between 25 and 35 years of age, and the patterns of age distribution as

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a whole are rather similar, such a substitution does not affect the total paternity rate very much. A series of tests showed that the error from this source does not exceed 5 per cent in most cases. The whole procedure corresponds exactly to the well-known methods of estimating total fertility rates, females being replaced by males in the calculation. It is at the same time much simpler than the technique described in an earlier article on the same subject[2] and has the advantage that a tabulation of the male population by occupation, age, and marital status in combination is no longer required.

Actually to compute fertility rates by relating the births to the total number of females in the corresponding age groups is not advisable. The concentration of gainfully employed women in a few occupations such as clerical work, semiskilled factory labor, and domestic service, would overweight some of the social-economic groups with unmarried females and make their total fertility rates appear unduly low. Another disturbing factor would be the presence of a large number of girls without gainful occupation. It is obvious that total paternity and fertility rates will be identical if there is no excess of either sex in the adult population.

Total paternity rates are independent of marriage frequency and marriage age. As fathers of illegitimate children are usually not reported, this part of reproduction has to be excluded from the rates. Even where illegitimate births are infrequent, their omission undoubtedly tends somewhat to understate social contrast in reproduction.

Total paternity rates can be used for the study of the present status of differential reproduction in England and Wales. Part IIa of the Registrar General's Decennial Supplement for 1931, which was issued last fall, is chiefly devoted to mortality, but it contains also the necessary basic data for the present objective. The male population is given by occupation and age in groups of 5 and 10

[2] Tietze, C.: The Measurements of Differential Reproduction by Paternity Rates. *Eugenics Review*, July, 1938, XXX, No. 2, pp. 101-107.

years, and legitimate births by occupation of father, but not by age of father.[3] Therefore the substitution method has to be applied. As a standard, the Australian age-specific paternity rates for 1933 were chosen. The Registrar General's social-economic grouping is primarily designed for mortality studies. The material has been regrouped for the present purpose into six groups which roughly correspond to the classification introduced into American statistics by Alba M. Edwards.[4] Each group covers as far as possible a man's whole occupational career from youth to ripe age. This is important for the following reason: Master artisans, shopkeepers, etc., as a rule do not marry before having reached economic independence, which is most often at about 30 years of age—at least in England. But if such an individual sets up a shop of his own earlier because his father has died and left him some money, or by another similar chance, he usually marries soon afterwards. The proportion of independent craftsmen, dealers, etc., below the age of 25 is very small, but a much greater proportion of these young men is already married than is the case in the population at large. The same thing may also be observed in the next age group, but in a less pronounced manner. These conditions tend to increase the number of births in the proprietary class and to reduce at the same time the paternity rates in those groups which contain a considerable number of "future proprietors." It is for this reason that within social-economic groups 2 and 6 in Table 1 employers, workers on their own, and wage earners are combined. In the professional class the non-inclusion of approximately 30,000 students in the official figures has been compensated for.

Table 1 presents the familiar pattern of an inverse relation between social status and fertility, but there are a few points worth

[3] The Registrar General's Decennial Supplement, England and Wales, 1931, Part IIa, Occupational Mortality; Tables 4A, 4D, and 14. Some additional information regarding age was taken from the Occupational Tables of the Census of England and Wales, 1931.

[4] Edwards, Alba M.: A Social-Economic Grouping of the Gainful Workers of the United States. Washington, United States Government Printing Office, 1938. 264 pp.

Social-Economic Group	Males 20-64	Legitimate Births	Total Paternity Rate
1. Professional Persons	328,955[1]	11,208	1,418
2. Proprietors, Managers, Officials, Clerks, and Kindred Workers	2,291,725	90,871	1,624
3. Skilled Workers	4,063,409	218,477	2,096
4. Semiskilled Workers	2,056,205	130,728	2,420
5. Unskilled Workers	1,806,980	119,016	2,657
6. Farmers and Farm Laborers	667,117	32,819	2,083
All Occupied and Retired Males	11,214,391[1]	603,119	2,134

[1] Including 30,000 students (estimate).

Table 1. Males, 20-64, legitimate births, and total paternity rate, by social-economic groups. England and Wales, 1931.

mentioning: More than a third of group 4 are coal miners. These are very fertile, even more so than the unskilled workers. Their total paternity rate is 2,761. The rest of the group—mostly semiskilled factory labor—has a much lower rate: 2,223. The total paternity rate of the agricultural group is remarkably low, which is partly due to late marriages and frequent celibacy. This situation is in striking contrast to that in the United States where the farm population is more fertile than any other of the large social-economic groups.

How many of these children survive their first year? Table 14 of the report gives infant mortality rates based on the deaths of 1930-1932 and the births of 1931 only. Table 2 shows a complete parallelism between total paternity and infant mortality rates. Infant mortality in the unskilled labor class is still more than twice as high as among the children of professional men, but as the percentages surviving their first birthday are 92.3 and 96.5, respectively, the difference is unimportant and the subject has lost very much of its former interest. The subtraction of infant deaths from total births does not affect materially the picture presented by the total paternity rates. These "effective" rates are also given in Table 2.

From the English life table for 1930-1932 it can be calculated with the help of the same Australian paternity rates as used above, that

Social-Economic Group	Infant Mortality	"Effective" Total Paternity Rate (Births Minus Infant Deaths)
1. Professional Persons	35.0	1,369
2. Proprietors, Managers, Officials, Clerks, and Kindred Workers	44.8	1,551
3. Skilled Workers	55.7	1,980
4. Semiskilled Workers	72.4[1]	2,245[2]
5. Unskilled Workers	77.2	2,452
6. Farmers and Farm Laborers	54.6	1,970
All Occupied and Retired Males	61.5	2,003

[1] Coal miners 82.8, rest 65.0.
[2] Coal miners 2,533, rest 2,078.

Table 2. Infant mortality and "effective" paternity rate, by social-economic groups. England and Wales, 1931.

a total paternity rate of 2,365 is necessary to replace a population under present mortality conditions, and that 2,219 children must survive their first year. It is obvious that only unskilled workers and miners surpass this standard, and these only a little. The rate for the professional group is almost 40 per cent below replacement value.

It should be remembered that illegitimate births are excluded from all these figures; they number not more than 4.6 per 1,000 legitimate births in the population as a whole. It seems just possible that their inclusion would raise the paternity rate of semiskilled workers other than miners to the replacement value.

The temptation is very great to compare the total paternity rate for each group in 1931 with the corresponding rates of an earlier year, say 1911. In view of the sweeping changes in occupational classification between these two censuses such a comparison should, however, not be attempted except in the broadest and most general terms. For this reason the data for 1911 are not given here in full. Total paternity rates at this earlier period ranged from about 2,000 in the professional class to 4,900 among miners. There has been a rapid decline of the birth rate in all social-economic groups from 1911 to 1931 and it has resulted in a narrowing down of the dif-

ference between the least and the most fertile section of the population. This corresponds closely with the findings of all other investigators in the field.

SUMMARY

For the study of differences in reproduction between social-economic groups it is recommended to refer births to total males instead of females. The computation of such "total paternity rates" by the substitution method is discussed. An analysis of the 600,000 legitimate births in England and Wales in 1931 reveals a marked inverse relationship between fertility and social status which is mitigated very little by differential infant mortality. Only unskilled laborers and miners produced sufficient children at this period to replace themselves.

TRENDS IN AGE-SPECIFIC FERTILITY RATES[1]

HENRY S. SHRYOCK, JR.

FERTILITY has been declining for over half a century in many of the countries of the Western World. As the culture pattern of family limitation diffuses throughout the populations of these countries, progressively fewer births are involuntary. It is conceivable that in the not so distant future of some of the more demographically "advanced" countries, only a very small residual of births will remain involuntary. The important question arises whether then their gross reproduction rates will continue to fall or whether they will be stabilized for a time below the replacement level but above zero. Until the family is relieved of some of the burdens of parenthood or until a new set of attitudes on the desirable family size develops, the gross rate may possibly fluctuate within a relatively narrow range about this level. The phase of the business cycle might well be the most important factor in these fluctuations.

Is there any evidence that such a period of stabilization has already begun in any areas or in any groups? If fertility stabilizes first in the more demographically "advanced" sectors, differentials between areas and different social and economic groups will be narrowed. There have been actually some instances of the narrowing of such differentials. Fertility, as measured by ratio of children to women of childbearing age, declined relatively more in the southern American states from 1920 to 1930 than in the Northeast, where the vital revolution first affected the United States.[2] Similarly from

[1] From the School of Public Affairs, Princeton University, in cooperation with the Milbank Memorial Fund.

[2] Space does not allow a discussion here of the alleged stabilization of fertility among native whites in New England, debated in this journal a few years ago. *See* Willcox, Walter F.: Changes Since 1900 in the Fertility of Native-White Wives. The Milbank Memorial Fund *Quarterly Bulletin*, July, 1932, x, No. 3, pp. 191-202, and Lorimer, Frank: Factors Influencing Ratios of Children to Native-White Women in the United States, 1900 and

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1910 to 1930 the fertility of native-white women declined 16 per cent while the fertilities of the more fertile foreign-born and Negro women were declining 30.2 per cent and 24.7 per cent, respectively.[3] Stouffer found that the margin of superiority in fertility of Catholic over non-Catholic wives decreased in Wisconsin cities during the period 1919 to 1930.[4] European studies have also revealed a few instances of the lessening of relative interclass differentials. However, all of these narrowings of differentials arose from divergent rates of decline in the fertility of the two groups compared and not from a rising or stabilized fertility in the group where the decline began earlier. The writer knows of no direct and conclusive evidence of the stabilization of fertility in any geographic region or social-economic class that has taken up the family limitation pattern. The absence of such instances may, of course, be due to the lack of proper data.

We do possess statistics on the birth rates of age groups, which are relatively comprehensive and comparable in time and space. The course of fertility in the different age groups has been by no means the same since the trends in these groups are partially independent. It would seem plausible, therefore, that a fixed level or plateau would be attained earlier by some age-specific rate than by the gross reproduction rate. Accordingly, it might be profitable to analyze these rates to determine at what ages fertility has decreased and is now decreasing most and least, the existence of increases or stabilizations in the rate, and similar points.

To consider first the United States, we may utilize data in the National Resources Committee's POPULATION STATISTICS. 2. STATE DATA. Table 1 of the present article presents from this source cor-

1930. "Preprint". *See* The Milbank Memorial Fund *Quarterly Bulletin*, July, 1933, xi, No. 3, pp. 240-241.

[3] National Resources Committee: THE PROBLEMS OF A CHANGING POPULATION. Washington, Government Printing Office, 1938, p. 127.

[4] Stouffer, Samuel A.: Trends in the Fertility of Catholics and Non-Catholics. *American Journal of Sociology*, Sept., 1935, xli, No. 2, pp. 143-166.

Dates	Age							Gross Reproduction Rate
	15–19	20–24	25–29	30–34	35–39	40–44	45–49	
1918–1921	51.7	159.6	157.4	121.0	83.0	33.2	4.0	148
1929–1931	47.2	130.8	125.5	91.4	58.5	22.8	2.4	116
Average Annual Percentage Change	−.9	−1.9	−2.1	−2.6	−3.3	−3.5	−4.7	−2.3

[1] Adapted from: National Resources Committee: Population Statistics. 2. State Data, Washington, Government Printing Office, 1937, p. 3.

Table 1. Corrected fertility rates per 1,000 native-white women in the United States, 1918-1921 and 1929-1931, and their average annual percentage changes.[1]

rected rates for native-white women in the United States, 1918-1921 and 1929-1931. These rates are partly estimated, but the Statistical Bureau of the Metropolitan Life Insurance Company has published[5] age-specific birth rates for white females in the expanding Birth Registration Area, for 1920-1924 and 1930-1934, that exhibited a very similar pattern of declines.

There is in both sets of data a clear-cut relationship between the percentage decline in the rate and the age of the women. The percentage decline increases consistently with advancing age. The average annual decline shown here of less than one per cent at ages 15-19 is markedly lower than that of almost five per cent at ages 45-49.

The National Resources Committee's table also gave rates for individual states. In thirteen states and the District of Columbia, the rate for women 15-19 actually increased over the period.[6] None of these was a southern state.

Let us see how similar to the American experience the trends in other countries have been. In order to secure some measure of

[5] Metropolitan Life Insurance Company: Decline in Birth Rate in Relation to Age of Mothers. *Statistical Bulletin*, December, 1937, xviii, No. 12, p. 5.

[6] Seven of these states were not in the Birth Registration Area in 1918 so that for at least some years their rates were estimated. Because of the War, fertility was somewhat unusual in the individual years of the earlier period; but the abnormalities tended to be mutually compensatory.

temporal and international comparability, the main analysis will be confined to the series in Table 11 of the STATISTICAL YEAR-BOOK OF THE LEAGUE OF NATIONS.[7] Table 2 is adapted from this source.

A systematic analysis of these data is not easy. There is a wide variety of dates and of total periods spanned. International comparisons thus become difficult. Many rates are averages of several years, making it impossible to graph annual fluctuations for the age-groups. Accordingly attention has to be restricted largely to relative changes at different ages in a given country over a given period. However, in order to introduce some degree of international and temporal comparability, the percentage changes have been reduced to an average annual basis. A rate is assumed to have changed geometrically (i.e., with a constant annual percentage change) between the mid-points of the dates that delimit the period being considered.

Table 3, then, presents these average annual percentage changes in age-specific fertility rates and in gross reproduction rates over two arbitrary periods. Within these periods, which coincide only approximately for the different countries, it is possible to make some qualitative comparisons as to patterns of change. The first period includes the whole range of post-War years, extending as far as possible in both directions to bring out the underlying secular trend. The second is the most recent part of the first but includes some countries for which data were not available for the complete range. It begins about 1934 and often represents a time of partial economic recovery. Here new patterns of change in age-specific fertility rates may represent either the beginnings of new secular trends or merely cyclical or annual variations about the old ones.

Countries are ranked in order of their gross reproduction rates at the beginning of each period. The post-War experience as shown

[7] Authorities may differ somewhat in the estimations often involved in calculating age-specific birth rates, particularly those for postcensal years. Hence it is not always safe to compare rates from different sources for different dates. The YEAR-BOOK's figures are not in every case the official ones. Not all of the rates are of equal reliability.

Table 2. Live births per 1,000 women of specified age, and gross reproduction rates.[1]

Country	Dates	Age							Gross Reproduction Rate
		15–19	20–24	25–29	30–34	35–39	40–44	45–49	
Sweden[2]	1921–1925	17.9	97.2	132.2	120.3	93.5	48.2	6.1	1,239
	1935	17.2	76.4	90.9	77.4	52.2	22.5	2.5	808
Switzerland	1934–1936	7.9	67.4	116.2	98.1	56.7	21.0	2.0	898
	1937	7.0	65.6	111.0	92.2	54.1	18.9	1.7	856
Norway	1921	12.3	106.4	171.4	161.5	132.1	71.7	12.5	1,632
	1934–1935	7.5	61.4	99.5	90.8	64.1	32.2	5.1	877
	1936	8.0	62.5	103.0	90.8	63.7	30.8	4.6	883
Estonia	1922–1926	9.7	87.2	133.5	112.0	82.4	37.1	5.4	1,133
	1935	12.3	81.7	104.3	89.9	60.4	26.0	2.6	907
	1936	12.3	86.7	108.7	88.5	62.2	25.8	2.8	945
France	1921–1922	25.3	134.7	151.7	103.7	61.6	23.4	2.2	1,226
	1934	33.3	125.7	122.9	82.9	46.0	16.7	1.4	1,051
	1935	27.4	123.6	118.9	78.5	43.7	15.6	1.4	1,003
Czechoslovakia	1921–1925	22.4	152.8	189.6	145.9	96.6	40.3	5.7	1,579
	1934	28.6	115.6	122.9	85.4	52.7	20.8	2.2	1,039
Denmark	1921–1925	23.8	120.3	158.7	131.8	92.1	39.7	3.9	1,387
	1934–1935	23.0	103.1	122.1	94.0	57.9	22.9	1.9	1,029
	1936	22.9	106.5	126.4	93.2	55.6	21.1	1.6	1,042
Latvia	1934–1936	13.5	82.0	118.5	102.0	68.9	29.1	4.5	1,014
	1937	14.0	98.1	122.8	103.6	66.3	25.8	2.9	1,056
New Zealand[3]	1921–1922	17.5	123.8	172.7	144.3	96.6	39.9	4.7	1,463
	1936	16.9	94.0	132.4	102.3	56.9	20.7	1.7	1,046
	1937	17.4	98.9	139.3	107.1	59.5	21.1	1.6	1,092
Hungary	1935	30.7	149.8	131.6	95.5	61.9	24.1	2.5	1,200
	1936	31.2	143.8	132.4	91.2	60.2	22.9	2.2	1,171
Finland[3 4]	1921–1922	14.1	120.3	164.7	150.1	128.4	73.8	12.2	1,569
	1930–1932	14.[illegible]	97.9	133.5	118.8	89.6	48.0	7.7	1,207
Netherlands	1935	11.1	81.0	147.5	138.0	99.1	43.3	4.3	1,262
	1936	10.7	81.7	146.3	137.0	96.5	42.8	4.2	1,256
Union of South Africa[3]	1924–1929	28.4	145.7	184.5	153.9	117.5	51.5	9.5	1,674
	1933–1934	30.1	132.7	157.3	126.6	91.4	41.6	7.3	1,427
	1936	32.3	143.1	163.2	130.3	84.1	36.5	6.3	1,448
Bulgaria	1921–1922	31.5	264.5	300.3	226.6	172.6	92.4	41.3	2,731
	1934–1935	52.6	199.0	193.2	128.6	78.6	36.4	11.8	1,696

[1] Adapted from: Statistical Year-Book of the League of Nations, 1937/38. Table 11, pp. 58–59.
[2] Age-specific rates represent confinements.
[3] White only.
[4] Based on *de jure* population.

Country	Dates	Age							Gross Reproduction Rate
		15–19	20–24	25–29	30–34	35–39	40–44	45–49	
		FIRST PERIOD							
Estonia	'22–'26 to '36	2.0	−0.1	−1.7	−2.0	−2.3	−3.0	−5.3	−1.5
France	'21–'22 to '35	0.6	−0.6	−1.8	−2.0	−2.5	−3.0	−3.3	−1.5
Sweden	'21–'25 to '35	−0.3	−2.0	−3.1	−3.6	−4.7	−6.1	−7.2	−3.5
Denmark	'21–'25 to '36	−0.3	−0.9	−1.7	−2.6	−3.8	−4.8	−6.6	−2.2
New Zealand	'21–'22 to '37	−0.04	−1.4	−1.4	−1.9	−3.1	−4.0	−6.7	−1.9
Finland	'21–'22 to '30–'32	0.4	−2.1	−2.2	−2.4	−3.7	−4.4	−4.7	−2.7
Czechoslovakia	'21–'25 to '34	2.2	−2.5	−3.9	−4.8	−5.4	−5.8	−8.3	−3.7
Norway	1921 to 1936	−2.8	−3.5	−3.3	−3.8	−4.7	−5.5	−6.4	−4.0
U. of S. Africa	'24–'29 to '36	1.4	−0.2	−1.3	−1.7	−3.5	−3.6	−4.2	−1.5
Bulgaria	'21–'22 to '34–'35	4.0	−2.2	−3.3	−4.3	−5.9	−6.9	−7.1	−3.6
		SECOND PERIOD							
Norway	'34–'35 to '36	4.4	1.2	2.3	0	−0.4	−2.9	− 6.6	0.5
Switzerland	'34–'36 to '37	− 5.9	−1.4	−2.3	−3.0	−2.3	−5.1	− 7.8	−2.4
Estonia	1935 to 1936	0	6.1	4.2	−1.6	3.0	−0.8	7.7	4.2
Latvia	'34–'36 to '37	1.8	9.4	1.8	0.8	−1.9	−5.8	−19.8	2.0
Denmark	'34–'35 to '36	− 0.3	2.2	2.3	−0.6	−2.7	−5.3	−10.8	0.9
New Zealand	1936 to 1937	3.0	5.2	5.2	4.7	4.6	1.9	− 5.9	4.4
France	1934 to 1935	−17.7	−1.7	−3.3	−5.3	−5.0	−6.6	0	−4.6
Hungary	1935 to 1936	1.6	−4.0	0.6	−4.5	−2.7	−5.0	−12.0	−2.4
Netherlands	1935 to 1936	−3.6	0.9	−0.8	−0.7	−2.6	−1.2	− 2.3	−0.5
U. of S. Africa	'33–'34 to '36	2.9	3.1	1.5	1.2	−3.3	−5.1	− 5.7	0.6

Table 3. Average annual percentage changes in age-specific fertility rates and in gross reproduction rates.

in this table contains little evidence that the rate of decline in the gross reproduction rate varied directly with the initial amount of the rate. Thus for this period, fertility did not appear to be declining faster in the areas where the incidence of the downward movement was relatively recent.

Over the long-time period the pattern of increasing percentage decline in fertility with advancing age was very consistent. It will be recalled that the same type of association occurred in the United States during the 'twenties. There were only two discrepancies, one each in Norway and New Zealand. In six out of the eleven countries the fertility of women 15-19 rose.[8] There were eleven instances,

[8] The increases in Estonia and Finland were not quite significant statistically. It is well to bear in mind in reading Table 3 that the rates for women 45-49 and sometimes for

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in eight countries, of the fertility of a younger age-group becoming higher than that of an older one.[9] This transposition was usually due to a more rapid decline in the fertility of the older women, not to a rise in that of the younger. It reflects the fact that the younger women were increasing their relative contributions to the gross reproduction rate.

That this direct association between relative fertility decline and age was not simply a post-War phenomenon is revealed in a table published by Kuczynski that includes the pre-War period.[10] For some of the countries the data go back as far as the last quarter of the nineteenth century. The percentage decline in fertility increased regularly with the age of the women in Denmark, Finland, Germany, Norway, and Sweden. There was only one exception each in the cases of France and Austria. Further east, in Bulgaria, there was no particular association between the relative change and age.[11] There were actual rises in the fertility of women 15-19 in Denmark, Germany, Norway, and Sweden and of women 20-24 in France and Norway.

During the most recent years the previously described general pattern of age-specific fertility change was not nearly so consistent. The difference may be due to the fact that the shorter the period the more likely it is to exhibit cyclical or random fluctuations and to obscure the underlying secular trend. This short phase is noteworthy because the gross reproduction rate actually rose in six countries. It is necessary to go back to the years just after the World War to locate another such upturn in fertility. A very plausible explana-

those 15-19 are based on a few births so that the sampling errors of percentage differences between two such rates are rather high.

[9] The age-groups involved were 15-19, which passed 40-44 or 45-49, and 20-24, which passed 25-29, 30-34, or 35-39.

[10] Kuczynski, Robert R.: THE MEASUREMENT OF POPULATION GROWTH. New York, Oxford University Press, 1936, pp. 122-124.

[11] Similarly, in Japan from 1925 to 1930 the changes in fertility did not seem to be a function of age. However, it will be recalled that by the post-War period, fertility decline in Bulgaria conformed to that in Western Europe.

tion of at least part of the rises in this period is that marriages and births were postponed in the worst years of the depression and took place when economic conditions improved somewhat. Part of the upturn may have been due to a new secular trend, but the possibility does not seem to be very great. Fertility in the Union of South Africa was still relatively high, and we should not expect the beginning of a stabilization to appear there. Whether the gross rate increased or decreased seemed to have little relation to the association between relative change and age. To generalize very roughly, fertility rose somewhat among women below 30 but continued to decline among the older women. It rose even more frequently at 20-24 and 25-29 than at 15-19.

Other data from the same source as Table 2 show that, over the post-War years for which data are available, there was a rather consistent direct association between percentage decline of fertility and age in Australia and Poland. Except for a relatively very large drop among women aged 20-24, the Italian trends from 1930-1932 to 1935 were also quite in the usual pattern.

A very interesting finding was made by McCormick and Glick in a recent study of age-specific fertility rates in Wisconsin from 1920 to 1935 concerning the effects of the depression upon them. To quote from their article:

> Between 1920 and 1930 women thirty-five years of age and over had experienced a marked loss of fertility, those of ages twenty to thirty-four had shown a moderate loss, and those below twenty years had made a slight gain. Under the stress of hard times this situation was reversed, it being the younger women who then placed the severest restrictions on child-bearing, while the pre-depression decline noted among the older women continued practically unchanged. As business improved in 1934 and 1935, there was an upturn in the fertility-rates of women under thirty-five, especially those of twenty-five to twenty-nine, but none in the older groups.[12]

[12] McCormick, Thomas C. and Glick, Paul C.: Fertility Rates in Wisconsin, 1920-35. *American Journal of Sociology*, November, 1938, xliv, No. 3, p. 404.

(Continued on page 302)

In order to see to what extent the development described by McCormick and Glick for Wisconsin took place elsewhere, the writer broke down the earlier part of the post-War range into two rough divisions. The first division may be termed the "pre-depression" period. It extended up to 1930. The second division could be called the "depression" period. It ran from about 1930 to about 1933 or 1934, births in the latter years usually representing conceptions in the trough of the world depression. It is realized that the course of economic activity varied somewhat from country to country, but no attempt was made to define national business cycles.

It is unfortunate that we had so few data that provided a comparison with the Wisconsin experience. Only Austria, Sweden, Norway, France, Czechoslovakia, Hungary, and the Union of South Africa had suitable rates for the depression phase. Briefly, very little parallelism could be found. Indeed, there were surprising rises during the depression in the fertility of French, Czechoslovakian, and Hungarian women aged 15-19. About the only similarity was furnished by Austria. This country has suffered chronic depression at practically all times since the War, but conditions began to get much worse about 1930 and reached their low point in 1933. Kuczynski's previously cited table reveals that from 1928 to 1931-1932, percentage fertility change varied with age in the usual way, there being even a rise among 15-19 year-old women. However, from 1931-1932 to 1933-1934 there was a very precipitate decline in the fertility of women 15-19 and the percentage decline decreased through 35-39, beyond which age group it increased.[18] Partial similarity to the Wisconsin and Austrian experience was exhibited by Czechoslovakia and Luxemburg. Before the depres-

Wisconsin was one of the thirteen American states previously mentioned whose fertility rate at ages 15-19 increased from 1918-1921 to 1929-1931.

[18] The percentage changes at each age were: 15-19, −14.4; 20-24, −6.3; 25-29, −4.0; 30-34, −3.8; 35-39, −3.5; 40-44, −4.4; 45-49, −5.1. It is interesting to note that, although Austria's gross reproduction rate was the lowest recorded for any country, the fertility rate at ages 15-19 has been higher than in most of the Western World.

sion and from 1930-1931 to 1932-1933 there was a direct association between Czechoslovakian fertility decline and age. From 1932-1933 to 1934, and in Luxemburg from 1930-1932 to 1935-1936, the declines were like those in Austria during the depression. It should be noted that the sequence of the patterns in these two countries corresponded only very roughly with the pre-depression and depression periods as previously defined. The fact that France and Sweden had the same type of association between fertility decline and age both before and during the depression makes it evident that this great catastrophe did not everywhere have the same kind of effect.

There was some tendency for the association between relative fertility change and age to be stronger in the pre-depression period than in the recovery phase. Here again the shorter average duration of the last period may be a partial explanation. In the pre-depression period relative fertility decline varied strictly with age in Sweden and Czechoslovakia and rather closely so in Estonia, France, Denmark, New Zealand, and Bulgaria. In all of these countries except Sweden and Denmark, the fertility of women aged 15-19 was increasing.

The data just described are not readily amenable to graphic presentation. From another source it is possible to show for Australia the annual movements in age-specific fertility rates from 1924 to 1935.[14] These rates are charted semi-logarithmically so that relative declines may be visualized. Here we see that annual fluctuations were small relative to the general trends. The same source also gave fertility rates for 1911 and 1921, and it is not surprising to find that the percentage decline in fertility from 1911 to 1935 varied uniformly directly with age. The usual pattern was also very well approximated throughout most portions of this range. (Figure 1.)

It may be contended that the observed variations of age-specific

[14] Wolstenholme, S. H.: The Future of the Australian Population. *Economic Record*, December, 1936, xii, No. 23, p. 197. These rates are for *female* births per 1,000 women. Hence they are slightly less than half the order of magnitude of the foregoing ones.

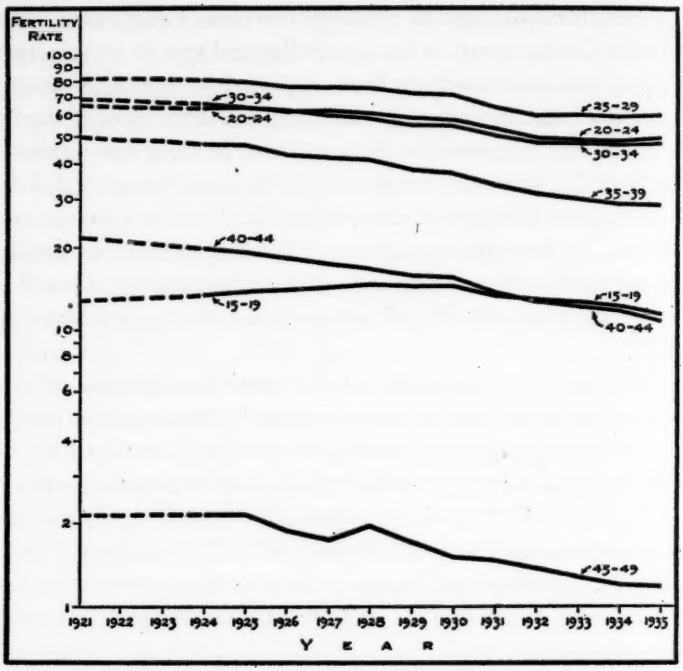

Fig. 1. Female births per 1,000 women by age-groups: Australia, 1921 and 1924-1935.

fertility changes were somehow largely a function of changing age-specific marriage rates. The effect of marriage may be examined directly through the course of age-specific marital fertility rates. Such data were available for three countries. The rates and their percentage changes are given in Table 4.

The figures for the United States are comparable with those abstracted in Table 1. The usual direct association between percentage decline and age is as consistent as before. The fact that the size of the percentage decline was greater for married women than for all women is in accord with the known decline of average age at marriage in the United States. (Illegitimate births are included in the

numerators of both types of rates.) We may conclude that changes in the proportion married at each age cannot account for either the fertility declines or for their incidence by age.

The Netherlands data are for 1906-1913 and 1935. Over this long period there was a rather good correspondence between percentage decline and age, except that there was little differentiation among ages 20-24, 25-29, and 30-34. The percentage loss in Italy from 1931 to 1936 decreased slightly up to age 30-34 and then rose regularly and sharply. (Curiously, there was no reflection in the marital fertility of the large drop in general fertility noted among women 20-24 from 1930-1932 to 1935.) Illegitimate births are not included for these two countries, but their distribution by age would have to have undergone an enormous shift to demolish the patterns found for legitimate fertility.

Although the evidence of just three countries is hardly conclu-

Table 4. Age-specific marital fertility rates and their percentage changes.[1]

Country	Dates	Age							
		10–14	15–19	20–24	25–29	30–34	35–39	40–44	45–49
U.S., Native-White	1918–1921	—	457.2	322.4	219.6	154.0	103.4	42.0	5.2
	1929–1931	—	408.1	258.5	160.0	112.4	71.4	28.4	3.1
Average Annual Percentage Change		—	−1.1	−2.1	−2.5	−3.0	−3.5	−3.7	−4.8
Italy	1931	209.9	397.3	355.2	267.9	205.6	151.2	71.3	8.6
	1936	191.6	370.6	331.9	259.2	189.5	133.3	61.5	6.4
Average Annual Percentage Change		−1.8	−1.4	−1.4	−0.6	−1.6	−2.5	−2.9	−6.7
Netherlands	1906–1913	—	745	479	372	286	219	106	12
	1935	—	533	308	239	185	126	56	6
Average Annual Percentage Change		—	−1.3	−1.7	−1.7	−1.7	−2.1	−2.5	−2.7

[1] Adapted from data in:
National Resources Committee: Population Statistics. 2. State Data. Washington, Government Printing Office, 1937, p. 4.
Italy. Istituto centrale di statistica: La diminuzione della fecondità legittima secondo l'età dal 1931 al 1936, *Notiziario demografico*, May, 1938, xi, No. 5, p. 88.
Netherlands. Centraal Bureau voor de Statistiek: *Bevolkingsstatistiek*, September, 1937, No. 5, p. 3.
United States figures are all live births, Italian figures legitimate live and still-births, and Netherlands figures legitimate live births.

sive, it seems highly probable that the general pattern in the age-incidence of fertility change rested largely upon a corresponding pattern for marital fertility. However, the frequent stability of the fertility of all women 15-19, going back in some cases into the nineteenth century, may well be partly the result of the gradual decline in average age at marriage in many parts of the Western World. It is rather striking that specific fertility rates of these women should vary so much among countries of similar culture and with similar gross reproduction rates. However, there were also wide differences in the proportion married at this age, and this factor was responsible for much of the variation. Different illegitimate fertility rates constituted another, but less important, part of the explanation.

SUMMARY

We may tentatively conclude that the pattern of percentage decline in fertility varying directly with age had been quite well established in the experience of the Western World, possibly as far back as the beginning of the vital revolution in the last quarter of the nineteenth century. The major component of this pattern was very likely a similar pattern for marital fertility. Had it not been for a secular trend toward earlier marriage, there would probably have been a general slight decline in the fertility of women 15-19 rather than frequent stable and upward trends. Very few countries can be found where the relationship between fertility decline and age was reversed during the depression, with decreases up to about age 30 being then relatively greater among the young women. Only future rates can tell whether the frequent rises in the fertility of women below 30 since about 1935 represent the onset of a cessation of fertility decline there or whether they are simply the result of the recovery phase in a particular business cycle. At present the latter alternative seems more likely.

It has been suggested that the fertility rates of women in the later part of the reproductive period have been dropping rapidly as an

indirect result of their past fertility performances but that such declines at these ages will prove to be temporary. The argument runs that these older women had large families when they were younger because they practiced but little family limitation. Later in life, when they took up family limitation, they tended to put an abrupt stop to childbearing. Younger women of more recent cohorts are becoming increasingly familiar with family limitation methods. When these younger women reach the older ages, they may still be planning pregnancies. Hence there may soon be an upturn in age-specific fertility rates above 30 years. Such a development would mean the future destruction of the general inverse association between fertility decline and age. Present trends afford no evidence of this upturn.

All in all, it seems probable that gross reproduction rates, even in demographically "advanced" countries, are due for still further declines before stabilization. The experience of the next few years may well clear up some of the questions raised here. Fortunately, age-specific rates are being published more frequently and for an expanding coverage.

ANNOTATIONS

NEGRO IMMIGRANTS OF TODAY

NEITHER in the many books on Negro problems in this country nor in the numerous studies of the immigration problem can one find more than passing mention of the Negro immigrant. Dr. Reid's recent study, THE NEGRO IMMIGRANT,[1] therefore, partially fills a distinct gap in the literature.

The author has set for himself the task of describing the background, characteristics, and social adjustments of foreign-born Negroes in this country during the period 1899-1937. During that period approximately 144,000 Negro aliens were admitted into this country and in 1930, according to the Census, there were approximately 100,000 foreign Negro residents within our borders. In comparison with the white immigrants during the period studied, the Negro immigrants, then, are numerically unimportant. As a part of the total Negro population of our country, too, the foreign-born Negroes can be said to constitute a minority within a minority. The author demonstrates well, however, that the numbers involved give an inadequate index of the importance of this migration, particularly in so far as the effect on native Negro culture is concerned.

The annual immigration reports use the term "Africans Black" as a cover-all designation of Negro immigrants. Actually, the bulk of these immigrants are not from Africa and neither are they pure blacks. They come mainly from the Caribbean area and represent a polyglot collection of racial mixtures. About three-fourths of the foreign-born Negro inhabitants of this country in 1930 were from the West Indies.

With respect to rural background, recency of migration, and large-city destination, the movement from the Caribbean area to this country co-

[1] Reid, Ira De A.: THE NEGRO IMMIGRANT. New York, Columbia University Press, 1939, 261 pp. $3.50.

incides roughly with our internal Negro migration. Less than one-tenth of the present Negro immigrant survivors arrived as early as 1900, as compared with one-third of the foreign white population. The Negro immigrants reside mainly in large cities of the Atlantic Coast. In 1930, 65 per cent of all foreign-born Negro residents in the United States were in New York City.

The author secured materials for his analysis of social adjustments from a variety of sources. With the aid of the Julius Rosenwald Fund he collected life stories, some of which are published in whole or in part in the present volume. He drew upon the files of the National Urban League for many unpublished documents. Those who know the author, however, will agree that perhaps his greatest asset for writing on this subtle subject is compounded of his own Negro ancestry, the bent of his interest, and his first-hand contacts acquired by a long period of residence in Harlem and extended affiliation with the New York Urban League and the National Urban League.

According to Dr. Reid, the Negro immigrant from the West Indies is not only "an alien by law and fact, but he suffers a complete change in status by emerging from a group setting where he was the racial majority into one where he becomes part of a racial minority." A new set of interracial problems is faced and he becomes acutely aware for the first time that his bronze-colored skin sharply delimits his economic and social opportunities.

Neither does the Negro immigrant find any welcome or mental solace in his initial neighborhood contacts in the colored community. As a parallel to the term "dago," the native Negroes apply to West Indians the epithets "monkey chaser," "Spic," and "Garveyite." Stereotyped attitudes are developed to the effect that the West Indian Negroes are too ambitious, too thrifty, and "not to be trusted." On the other hand, the newly-arrived West Indian Negro develops the defenses that the native Negro's own unenviable position is proof of shiftlessness, laziness, and passive acceptance of discriminative practices. Reared, perhaps, in the Episcopal service, the West Indian looks down his nose at the illiterate and emotional character of much of the religious life in Harlem.

The author feels, however, that such personal conflict is but an incidental part of the process of accommodation and adjustment. He believes that the commingling of native and foreign Negroes in urban centers has already brought salutary changes in Negro-white relations. The West

Indians have contributed more than their proportionate share to Negro leadership and to the Negro professional group. To this extent they have helped to shatter the bases for the slave attitudes of whites toward Negroes. If the "ambitious" and "acquisitive" characteristics of the West Indians are often resented by native Negroes, they also provide a stimulus to competition. On the other hand, if the West Indian is initially disdainful of the native Negroes, he soon learns that his own fate is intimately connected with theirs. In time, the outcome should be an invigorated Negro unity. If the Negro immigrant, in seeking improvement in economic, political, and social status, "steps on the heels of the native Negro population, it is only because both groups hear the same drummer and are aligning in a common cause."

CLYDE V. KISER

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EDUCATING FOR HEALTH[1]

IN his preface Mr. Hill states that his aim is not a study of health education as an aspect of health work, but an over-all review of that activity as a part of the whole national movement for adult teaching and learning.

Such a general summary, provided it is well done, can serve both the health educator and the adult educator in other fields. For while we have excellent monographs and papers on popular health education and many good books on school health education, this is one of few books trying to indicate the important interrelationships of the health education field.

How successful has Mr. Hill been in attaining his objective? The author came to his task with no previous experience in the ways of the world of health. The gathering of the material took him into a dozen states and to many more cities and counties. What he lacked in health education background, he made up by a firm knowledge of other forms of adult education, great objectivity of approach, scientific attitude of mind, and a sense of words.

In ten chapters, the author traces the vast health educational move-

[1] Hill, Ernest Frank: EDUCATING FOR HEALTH. A Study Of Programs For Adults. New York, American Association for Adult Education, 1939, 224 pp. $1.25.

ment and the important elements which give it life: the origin of the American Public Health Association and its educational program and work; the heirs of Hippocrates and what the American Medical Association has done and does in the way of health teaching; the crusades of voluntary health agencies; the educational activities of the United States Public Health Service, of state and city health departments and of commercial organizations; the work of the foundations that have endowed experimentation in health; the three health demonstrations financed by the Milbank Memorial Fund; the teaching of health education in colleges; nurses as teachers; and literature, radio, and movies as employed by health educators.

These subjects are treated selectively, with outstanding examples in each field, rather than comprehensively. The treatment is no mere paean of praise, but a discriminating evaluation. Even if some exceptions may be taken to certain statements made by the author, his opinions always deserve serious consideration.

Mr. Hill speaks with confidence regarding the quality of the technical work of the American Public Health Association and agrees that the place of the Association in education for health is unique. At the same time he points out that the great range of its work has given a "halting character to some of its activities," that there is justice for the complaints of some members that its journal is "ill-adapted to the needs of most health workers," and that the Association has not dramatized itself sufficiently. But Mr. Hill rightly adds that "in comparison with its positive contributions the faults of the Association are minor."

The author pays tribute to the health education activities of the medical profession and particularly of the American Medical Association. At the same time he feels that the Association has done little in issuing material on the educational factors involved in a physician's work, with parents of school children, with teachers and with public health nurses. He feels that much more than medical knowledge is needed for successful health education work; information about social conditions, health agencies, and about education methods should be important to the doctor. "The idea," says Mr. Hill, "that in dealing with nurses, teachers, parents, and even patients, they must be teachers as well as doctors is simply not known or accepted by a large number of physicians. To an outsider looking into the health field there is something preposterous about this situation."

To this statement medical educators would probably reply that medical colleges now take more and more cognizance of such needs and that the medical profession is devoting increasing attention to the problems of health education. Furthermore, as Mr. Hill himself points out, Dr. W. W. Bauer, director of the Bureau of Health Education of the American Medical Association, is co-author of a recent volume, HEALTH EDUCATION OF THE PUBLIC, which deals with methods of instructing the masses. The public health nurses receive considerable attention in this book, and the work of the National Organization for Public Health Nursing is sympathetically and accurately described.

Here are some of Mr. Hill's criticisms about health education as at present conducted. He thinks that there is too much dogma and propaganda and too little clarifying explanation in education for health. He finds that much of the printed material of health agencies is not well prepared and that in order to accomplish its purpose, it needs a happy combination of journalistic experience, artistic ability, and technical accuracy. All experienced educators and health officers will agree with Mr. Hill's statement, that where care, skill, and energy have been expended upon such material it has been highly effective.

Radio and visual education, he feels, have been even more neglected than printed materials. While some organizations, he writes, have put out very good health films, and others are conducting excellent radio programs, on the whole, health agencies have done little with films and broadcasts.

There can only be general agreement with his conclusion that to accomplish better results there is a special need of greater financial resources and of more facilities for training health educators of all types. "With increasing facilities will come improvement in quality and more opportunity for specialization."

One could quarrel with Mr. Hill concerning his assignments of space to some phases of the subject. Why, to give one illustration, only a half-page to the health work of the Children's Bureau? But, on the whole, Mr. Hill has compressed into this well-written book much valuable information concerning the health education movement. He has also managed to convey in his narrative something of the romance of the public health pioneers who reshaped the contours of life. It is a volume of high quality and holds our interest to the last page.

SAVEL ZIMAND

GUIDE BOOK FOR NUTRITION SURVEY

In his monograph on investigations into the nutrition of populations, Dr. Bigwood has set forth what he considers to be the guiding principles of a comprehensive survey.[1] Two approaches he portrays: inquiry into food consumption and examination of the nutritive state. Although each has been used singly, seldom have they been used conjointly. But this is understandable, says the author, since one or the other satisfies such varied objectives of information as national food supply, food consumption by a group, food expenditures, assessment of nutrition, and numerous physiological problems.

In Part I are described four types of dietary survey, classified by the nature and size of the social unit to be investigated: nation; specific social groups and institutions (army, prison, school), more or less homogeneous; family; and individual. The several techniques, appropriate to particular objectives and types of survey, are then considered in two steps: collection of the data and analysis. Under methods of collection, in turn, detailed instruction is given in three methods: weighing; records of purchases in household books; questionnaire. In conjunction with analysis of data, considerable preparative information is given in the science of diet covering such matters as the essential nutritive principles. Especially the vitamins are discussed at some length with consideration of their content in foods and their calculation in units. For the analysis of intake he lists two methods: direct, through chemical examination of foods consumed, used in physiological studies; indirect, through recourse to tables of composition, used in mass studies.

Even while the favorable points of the several techniques are indicated, the limitations are not neglected. Ranging in refinement and precision the methods of collection have definite limitations; consequently difficulties arise from applying a method to a purpose for which it is unsuited. Where the more sensitive method is used, the task may become so magnitudinous as to be impossible; *per contra,* if the least sensitive is used, the results may become so questionable as to be unacceptable. For example, employment of either the weighing or household book method becomes, as Dr. Bigwood points out, a formidable feat when individual intake is under investigation. Of the questionnaire method he is exceed-

[1] Bigwood, E. J.: GUIDING PRINCIPLES FOR STUDIES ON THE NUTRITION OF POPULATIONS. Geneva (Series of League of Nations Publications), 1939.

ingly critical, regarding it as a means of "fragmentary and very rough information" about dietary habits, with little promise of data quantitative in nature. Indeed, he dismisses it so shortly that he appears to have mentioned it only for the sake of completeness. Nevertheless, it is capable of greater adaptability and quantitation than is indicated in the monograph; properly modified it is, for certain purposes, to be neither despised nor rejected.

For all methods of collection, he recommends a week's duration, indeed emphasizing the point in a separate chapter with an adjuration in italics. In this he follows precedent, for the week-period has been handed on to the point of becoming sacrosanct. Although the supporting argument, based on criteria of sampling, holds for many cases, there is little or no evidence that the error in a week's inquiry is the minimum permissible under all circumstances. To pronounce doubt on any food intake records of less than a week's length is somewhat sweeping and a trifle on the dogmatic side, unless the purpose of the survey is specified. All in all, it is clear that in the collection of diet information, the author favors the weighing method for a week's duration with use of an investigator in the home. But that is time-consuming, laborious, and expensive. If information is sought on individuals, this approach becomes almost prohibitive for routine use. In the domain of public health at least, it raises the question, as yet unanswered, how much precision is necessary; and how can interests of precision be harmonized with limited time, personnel and funds. These very practical considerations are more than guiding principles in public health work; they are inexorable specifications.

In certain steps of the analysis also, the author points out limitations. Family surveys do not show how "the total food consumed by the family is divided among the different members." Consequently, if the results obtained for different families of dissimilar composition are to be compared, or if information is sought on per capita consumption, scales of family consumption coefficients are necessary. But even these only indicate how the family supply of the different nutrients should be, not how they are divided among the members. In McHenry's recent study, the mothers fared worst in the division. As another instance of limitations in technique the author emphasized the point, often overlooked, that the values in tables of food composition are approximate.

In Part II, devoted to a discussion of the methods of inquiry into the state of nutrition, the available tests are grouped under three headings:

clinical, somatometric, and physiological. This section enumerates the individual tests, elaborates on them with description of their purpose and nature, and outlines how they may be incorporated into three types of inquiry. Depending on the number and kinds of tests, these types form a progressive series in expansiveness and complexity, the more comprehensive type including all the tests of the simpler type, together with additional tests.

In matter and form the monograph displays both scholastic and literary workmanship. True, the inclusion of a section on statistical methods running to eighteen pages, while understandable, is perhaps superfluous. Characterizing pellagra as a secondary (endogenous) deficiency disease and linking it with parasitic infections needs a certain revision. Except for one or two instances, the arrangement of the bulk of the material is a model of coherence. For some unfathomable reason, the section on analysis of dietary data precedes that on methods of collection; and perhaps some of the paragraphs on deficiency states occurring in Part I might better appear in their more natural setting of Part II. But against the overwhelming excellence of the book on all points, these comments are so insignificant as to suggest cavil where none is intended.

Encyclopedic in nature, this monograph is a survey of methods with critical comment rather than a critique on rigidly selected methods. Prior to a revision and expansion, Part II originally appeared in the League of Nations ***Bulletin;*** but it is useful to have all the material within one cover. The contents are of interest for what some believe to be the next strategic and necessary step forward in nutrition. Bearing upon it, one statement takes on significance: "Only combined research covering both food consumption and the physical state of nutrition can . . . . define the immediate medical consequences of any slight departure from the standard thus established—in a word, to throw light on the problem of latent pre-deficiencies and their early diagnosis." It is not to be expected that the book gives the answer to this practical and basic point; nor to the urgent need, implicit in it, for a simple, economical, standardized methodology of precision for case detection of unsatisfactory nutrition. Certainly the various types of inquiries, cited in the book, do not represent the final word. Rather the book's merit is in exhibiting samples of technical ore out of which, it is hoped, a refined methodology may ultimately be forged. It belongs on the list of recommended reading in the field of public health.

H. D. KRUSE, M.D.